The Journal
OF THE KANSAS MEDICAL SOCIETY

Fifth Annual University of Kansas

School of Medicine Number

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March, 1951 Vol. LII, No. 3

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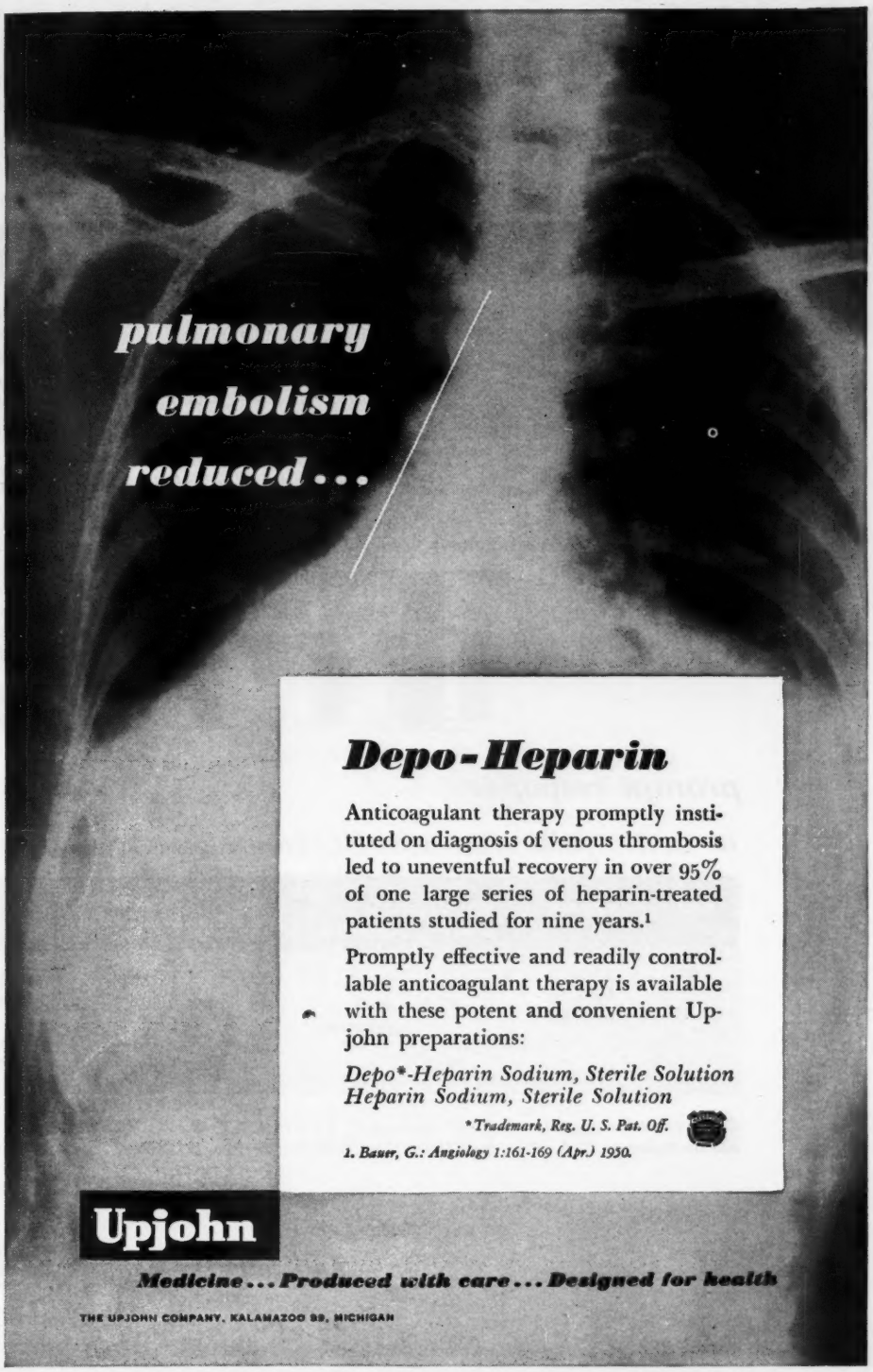
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		SOURCE	ORGANISM			
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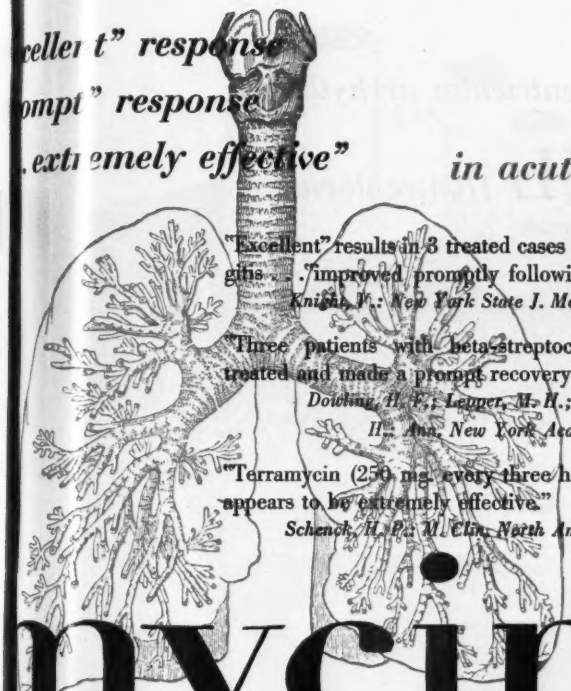

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Knight, V.: New York State J. Med. 50:2173 (Sept. 15) 1950.

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Dowling, H. F.; Lepper, M. H.; Caldwell, E. R., and Spies, H.: Ann. New York Acad. Sc. 53:433 (Sept.) 1950.

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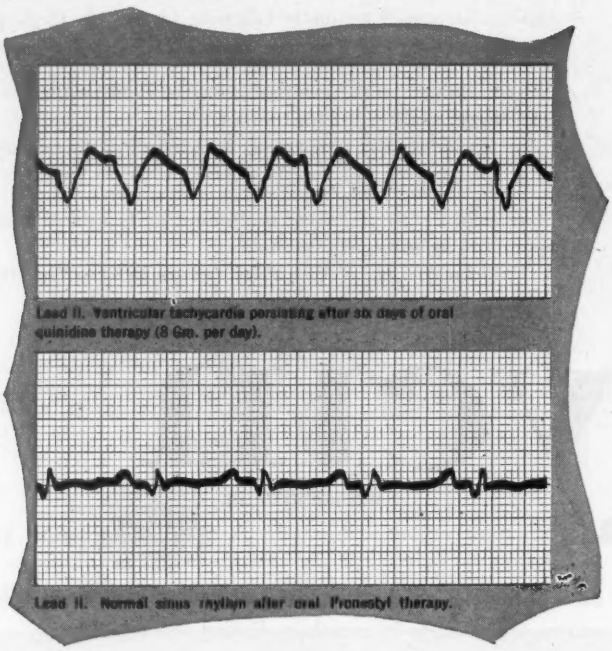

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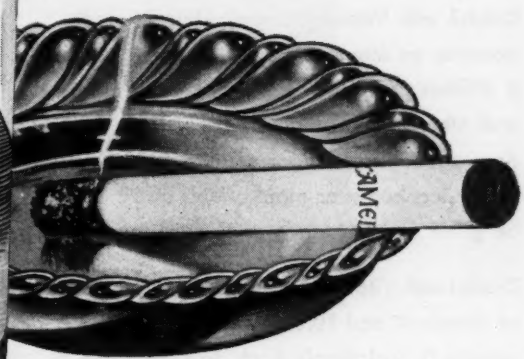

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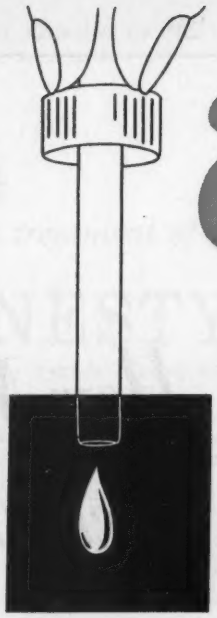

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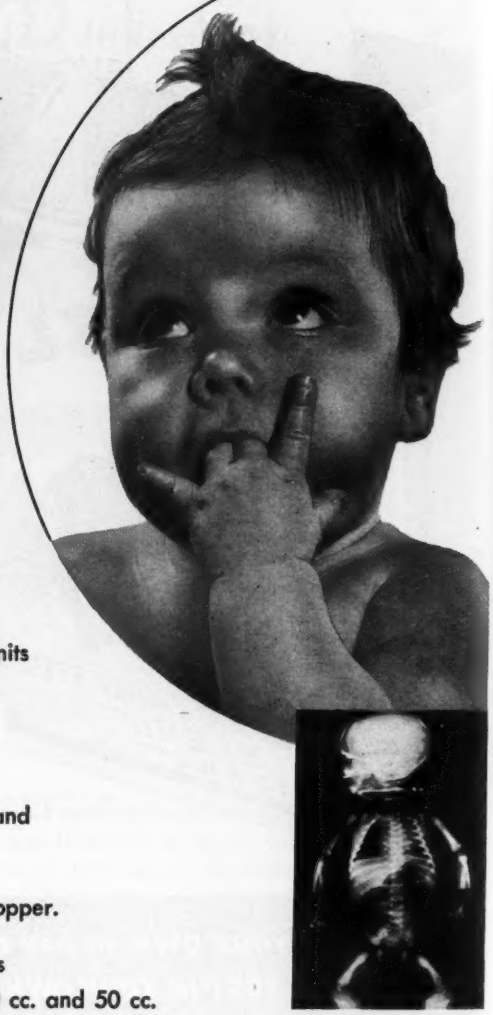

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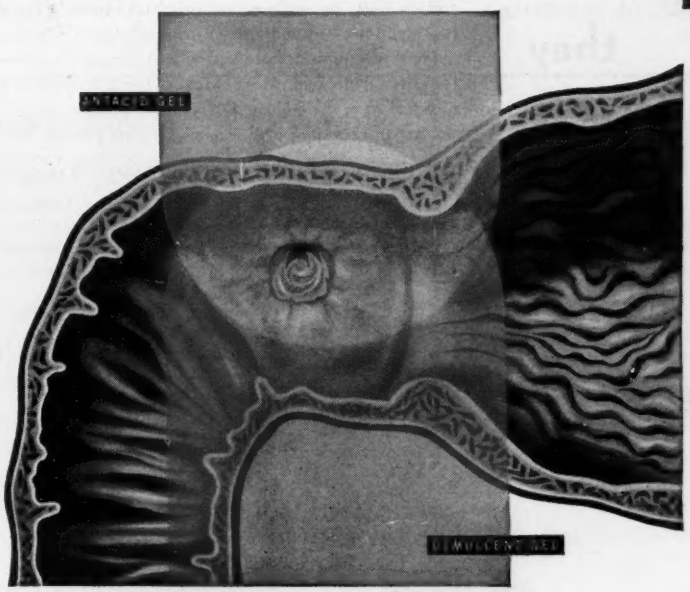

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References

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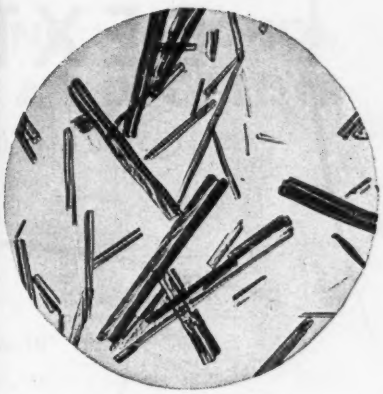

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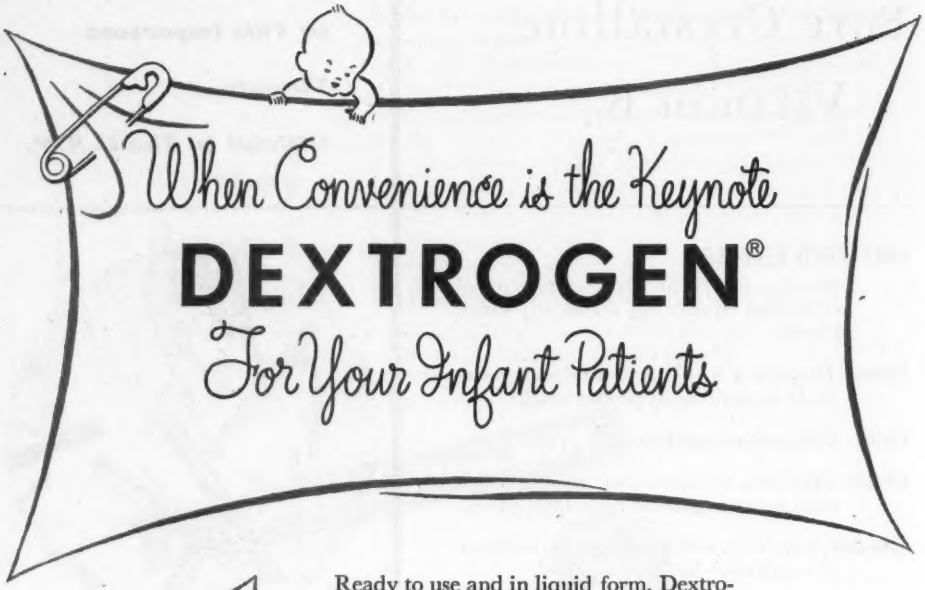

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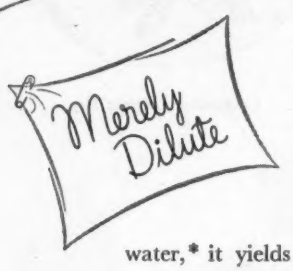

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Trochanteric Fractures of the Femur*

Experiences at the University of Kansas Medical Center with a Group of 45 Cases Treated by Non-operative Methods During the Past Five Years**

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Kansas City, Kansas

Trochanteric fractures of the femur are a common major injury, usually occurring in an aged individual as the result of a relatively minor trauma. The term is used here to include all fractures from the capsular attachment proximally to the immediate subtrochanteric area distally. It does not include isolated fractures of either the greater or the lesser trochanter, or subtrochanteric fractures without involvement of the lesser trochanter.

These fractures present at this time a problem of selection of proper treatment which was not present prior to the popularization of internal fixation, the availability of relatively inexpensive antibiotics, the general use of blood transfusions, and the shortage of hospital beds. It is commonly known that fractures in this area are extremely apt to unite, in contrast to intracapsular fractures, that they have a tendency to do so in a position of coxa vara and that they present a definite threat to the life of an elderly individual sustaining one. It is also commonly recognized that non-operative treatment, varying from simple recumbency with sand bags to prevent motion of the leg and to relieve pain, up through various forms of traction and even immobilization in a plaster spica, will often result in an extremity on which weight can be placed, if the patient survives the fracture.

During the last decade internal fixation of these fractures has become commonplace and published results indicate a considerable reduction in mortality coincident with this. It is not our purpose here to review the literature pertaining to this decreased mortality, it having been well summarized by Evans[1] and by Murray and Frew.[2] It is apparent from the literature that internal fixation of these fractures is not free from complication and does not always result in a hip with good function, Bickel and Jackson[3] having reported a mortality of eight per cent in a series of 61 operatively treated fractures who were traced, and "poor result" percentage of five per cent in this same series. They report a "poor result" percentage of zero in 26 fractures treated by skin traction, but a mortality of 31 per cent in this group. Since it is difficult to avoid an element of selection in operative series, it is difficult to be certain whether all of this apparent decreased mortality is due to the surgical treatment.

At this hospital there is no routine definitive treatment for trochanteric fractures of the femur. On admission a patient with this type of fracture is placed in Russell's[4] traction immediately and given a thorough work up from the standpoint of history, physical examination, and indicated laboratory procedures. This latter includes urinalysis, complete blood count, and blood chemistries as indicated, including at least an NPN. Medical management is started immediately to correct, insofar as possible, obvious complicating conditions such as dehydration, anemia, cardiac decompensation, cystitis, etc., and as soon as the work-up is complete, decision is made as to whether the patient should be treated by operative or non-operative means.

This decision may require evaluation of a portable x-ray made after 24 hours or more in Russell's traction, and a bedside evaluation of the patient's physical and mental condition after having been in traction for a few days. Should it be decided that the fracture should be treated surgically, this treatment is scheduled at a time when the patient has been put in the best possible condition for operation. Consultations are obtained as indicated from other services, particularly the medical and urological, both as to evaluation of the risks and as to treatment, but

*From the Orthopedic Section of the Surgical Service, University of Kansas Medical Center.

**A report of experiences with a similar group of fractures treated operatively is in process of preparation.

the details of treatment are usually carried out by the orthopedic section. Surgical treatment, if selected, is usually an open reduction with fixation with a Smith Peterson nail with a plate. Non-operative treatment consists usually of continuation of treatment in Russell's traction, although occasionally treatment with a spica cast or a Percy Jones type of well leg traction has been used.

There has been a tendency here to operate on an increasing number of these fractures, especially since facilities have been improved for so doing by completion of the new operating suite with an x-ray fluoroscopic table in the orthopedic room, but we still treat some patients in Russell's. This study was undertaken to attempt to evaluate and compare each method. Since many of our operative cases are too recent for adequate follow-up studies and evaluation, this report is confined to 45 non-operatively treated cases, treated here and obtained from the diagnostic index from 1945 through February, 1950.

Method of Study

The records and x-rays of these 45 patients were obtained and carefully reviewed. The fractures were classified according to Bickel and Jackson.[3] This classification considers a non-comminuted fracture across the intertrochanteric areas as a Group I (Figure 1), the same plus some comminution in the region of the lesser trochanter as a Group II (Figure 2), and those with marked comminution and usually with displacement as Group III (Figures 3, 4, 5). Bickel and Jackson include in this classification the so called "reverse intertrochanteric fracture" as described by Wright.[5] There were no reverse intertrochanteric fractures in this group of 45 fractures treated non-operatively.

Two additional subvarieties of comminuted fractures were noted in this series (Figure 5). In one patient there was more comminution in the region of the greater trochanter than elsewhere, and th fracture tended to assume a valgus position an healed in coxa valga. In three patients a lon spicule of bone attached to one of the comminute fragments was detached from the subtrochanteri region of the upper femoral shaft. The significanc if any, of this latter difference in fracture patholog is not known, the number of cases of this type small, and no special classification has been used fc these fractures.

Following review of the records and x-rays, th pertinent follow-up records were inspected. Eac surviving patient was then sent a questionnaire t fill out and return. In some patients who could nc be reached this way, the family physician or loc welfare office was asked for and supplied informa tion concerning the patient's status.

Results were classified as follows:

Excellent—Walks without assistance and without pain and limp, and has resumed former activities, fracture healed in anatomic position.

Good—No impairment of activities insofar as hip is concerned, walks without assistance on smooth surfaces and without pain. Some of this group had slight limp, coxa vara, and limitation of hip motion.

Fair—Solid union of fracture and could get about as could the "good" group insofar as hip was concerned, but had some pain in the hip and hence could be considered as incapacitated to varying degrees.

Poor—Could not get about at all without assistance, such as holding on to chair or using crutches, and grossly incapacitated because of hip.

Satisfactory—Fracture healing apparently satisfactory but other troubles prevented activities.

Fatal—Those who died prior to obtaining union

Figure 1. Group I fracture sustained, age 76. Treated by 41 days in traction. Result graded "good" 57 months after fracture.

Figure 2. Group II fracture sustained, age 64. Treated by 37 days in Russell's traction with crutches allowed 10 days later. Resumed full weight-bearing in six months and can walk three miles without pain, but with a slight limp. Graded "good" 42 months after fracture.

of their fractures or prior to dismissal from the hospital, whichever occurred last.

Clinical Analysis of Group

These patients as a group had passed their period of best health, to put it mildly, and many were in what would ordinarily be considered poor condition either for surgery or prolonged bed rest on admission, because of conditions antedating their trauma. Only nine of the group failed to present complicating factors on admission, the factors including deafness, blindness, old cerebral accidents, anemia, cholecystitis, diabetes, cardiac pathology, Parkinson's disease, leutic aneurysm, senility, and other degenerative conditions common to this age group. Several had osteoarthritis of the hip antedating the fracture. All presented difficult medical and nursing problems in varying degrees. Many were apprehensive as to their ultimate survival. Most of the patients had been injured by a fall in their own homes or yards, but a few were injured by falling at work, and in a few cases the exact mechanism of injury could not be determined because of the patient's mental condition and lack of adequate anamnesis from other sources.

In these 45 patients the average age was 77.6 years, the youngest being 50 years of age, the oldest 94. The fracture was on the left side in 22 and on the right side in 23 patients. Thirteen of the patients were males, and 32 were females, including three colored females. The type fracture was placed in Group I in 11 patients, Group II in seven, and Group III in 27. Some difficulty was encountered in classifying a few of these cases, since it is our experience from operative cases that many of these fractures are more comminuted actually than the x-rays would indicate.

Treatment

All of this group were treated in skin traction, and this was the only method employed in 42 cases. In one each of the remaining three cases Percy Jones well leg traction, immobilization in a plaster spica, and skeletal traction, respectively, were used after

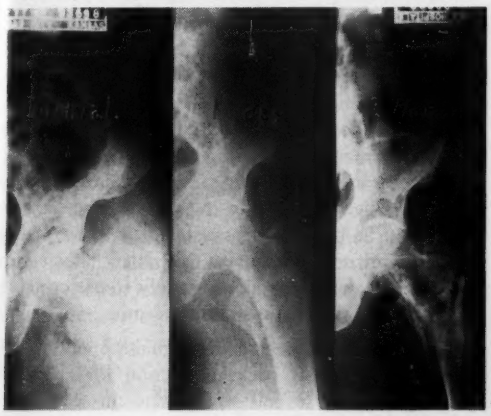

Figure 3. Group III fracture sustained at age 83. Treated in Russell's traction for 47 days. No complications in hospital and went home on 50th day. She resumed weight-bearing four months after injury and now walks without limp. Can go up and down stairs but does not leave house because of infirmity. Graded "good" 42 months after fracture.

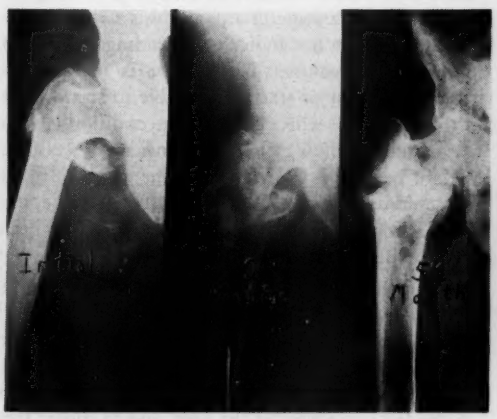

Figure 4. Group III fracture sustained at age 68. Treated by 63 days in Russell's traction. Some coxa vara developed when traction was removed, but patient was bearing full weight on leg in four months. Walks with a little limp which she attributes to arthritis of the opposite hip, and does all housework as before injury. Graded "good" 38 months after injury.

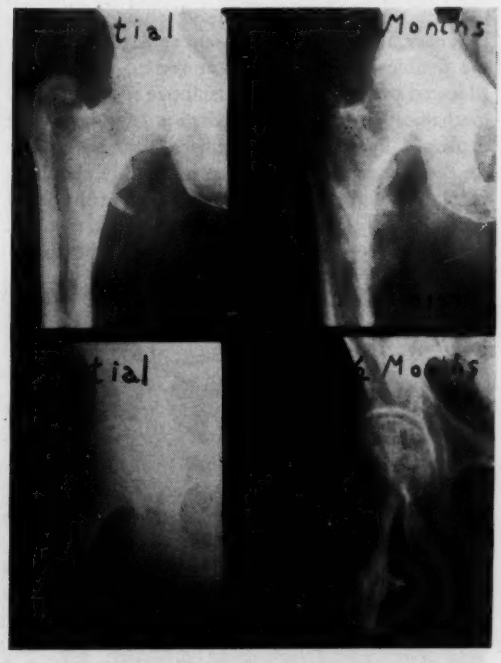

Figure 5. Upper—Group III sustained at age 79. Treated 41 days in Russell's traction. Is on her feet six hours per day and has resumed all housework. Graded "excellent" at 10 months after fracture.

Lower—Group III sustained at age 86. Had "valgus" tendency. Treated by 45 days in Russell's traction. Full weight-bearing started at 81 days. Does all housework as before fracture without limp or pain. Graded "excellent" 38 months after injury.

initial treatment with skin traction. In five patients, Buck's extention was employed, and in 40 Russell's traction was used (Figure 6). Counter traction was supplied by elevation of the foot of the bed. A trapeze has been routinely supplied to permit the patient to assist in moving for nursing purposes. When difficulty has been encountered with the patient sliding down in bed, thus allowing the Russell's footpiece to come in contact with the foot of the bed, a padded wooden box placed between the foot of the bed and the opposite foot has been found useful. Traction here has been applied to the skin by means of reinforced flannel strips, one on each side, applied to the skin with tincture of benzoin or "Ace" adherent, and kept in approximation by cotton elastic three-inch bandages. The areas about the head of the fibula, the patella, and each malleolus are well padded. The traction is checked at least twice daily by members of the orthopedic section, in addition to the usual observations by members of the nursing staff.

Usually eight pounds of weight will reduce these fractures adequately, and no sand bags, pillows, or rotating straps are necessary. However, we do not hesitate to apply more weight or to use these supplementary devices to control rotation if it is necessary. The usual fracture will reduce nicely in traction in the neutral position or slight abduction. However, if difficulty is encountered, traction with the double Russell's pulley moved to the slightly adducted position will often facilitate reduction. The mechanics of this seeming paradox have been well explained by Fox and Aphelbach,[6] but the reason for the success of the adducted position in obtaining reduction of the coxa vara may be more simply, if less accurately, understood by considering that this position reduces the amount of pull from the powerful adductor muscles, which tend to tighten up when irritation about the hip is present.

Figure 6. A patient in Russell's traction.

The progress of the fracture is checked daily by means of portable x-rays until position and alignment are judged to be satisfactory. This is usually obtained at the first portable x-ray 24 hours after admission. The absence of gross displacement and the presence of an angle of 130 degrees or slightly greater between the neck and shaft of the femur have been taken as constituting a satisfactory reduction. X-rays are then usually repeated at weekly intervals. The decision as to the length of time that the traction is to be left on is made on the basis of healing as demonstrated by x-rays and is also guided somewhat empirically by past experience as to what time would be adequate for a fracture belonging to that group.

When it is decided that the fracture has healed sufficiently, the patient is lifted out of bed into a wheel chair for a period of one hour twice per day, the traction being unhooked and resumed when the patient is in bed. After a few days of this, the films are repeated, usually sending the patient down to x-ray where a "bucky" film can be obtained. Should there have been no change in position, and healing appears to be progressing satisfactorily, the traction is then permanently removed, the patient allowed up in a wheelchair as desired, and taught to use crutches if the general physical condition permits.

Should a major degree of angulation occur, the patient may be put back in traction for an additional period of several weeks, but this has been found necessary in only three patients in this service. Lesser degrees of angulation occurred in 14 patients in this series at this point in treatment, but the matter was disregarded since a few degrees of coxa vara had not been found to be disabling to the average patient in this age group, and this slight change in position does not indicate impending non-union in this type fracture. The patient is usually dismissed when out of traction and able to spend most of the day in the wheel chair if the condition is such that crutch walking is not considered feasible. Weight bearing is deferred until the x-ray indicates that union has progressed to where it can be tolerated. Ischial weight bearing braces have not been used in this series.

Treatment of this type of fracture in Russell's traction is a laborious method and requires detailed supervision, not only of the traction but of the patient. The traction must be carefully watched to be sure that it does not slip and cause pressure sores on the leg or dorsum of the foot, and to insure that it is remaining effective. The patient must be kept in fluid balance, meticulous back care must be carried out to prevent decubiti from developing, if pos-

sible, or to heal them when small if they do develop. The patient needs reassurance that this period of bed rest in traction is compatible with survival and ultimate use of the leg. Ample medication for relief of pain must be given early so that the patient will move about enough to permit back care, but not so much that he will be lethargic and thus difficult to feed or more liable to incur respiratory infections. We believe that barbiturates are in general contraindicated in these patients.

The advantages of the use of an indwelling catheter to facilitate nursing care and prevent the bed from getting wet in elderly patients with poor bladder control must be weighed against the disadvantages of possible urethral or bladder infections possibly resulting from the catheter. The constipating effect of opiates, change of diet, and lack of activity must be considered and measures taken to prevent fecal impaction, and to recognize and treat it promptly if it occurs.

Infections, particularly pneumonia, must be closely watched for and energetically treated. Mental aberrations of a minor nature, tending to improve as the pain of the fracture becomes less during the first week and the patient gets adjusted to the new environment, need cause no concern, but progressive deterioration along these lines, especially if the patient is violent and persists in removing the traction, should indicate need for reevaluation of the patient and probably selection of some other method of treatment.

Results of Treatment

Of these 45 patients, nine died in the hospital, eight of these before obtaining union of the fracture, a mortality rate of 20 per cent. The average age of these nine patients was 84.1 years, and average period of hospitalization was 37 days. As a survey of Table I will show, the majority of these patients were in extremely poor condition even in comparison with other patients in this series, and several were never in condition to operate on had this been desired. Consequently, we do not feel that this figure represents a true picture of the hazards of treatment in Russell's traction in comparison to that of operative treatment. Even so, it compares fairly favorably with several reported operative series reported in the literature.[2] It might be questioned whether, since we are not yet reporting our operative group, we have kept these figures at low levels by doing late internal fixation on patients who had gotten into trouble in Russell's, but we know of no case during this time interval where the decision to operate was made after the eighth hospital day, and the average time from admission until surgery for the operative cases treated during this period has been five days.

Table I. Summary of Patients Who Died While Being Treated in Traction

Age	Type fracture	Treatment	Days of survival after injury	General health before fracture	Complications noted on admission	Cause of Death	Autopsy	Remarks
82	III	Buck's traction	47	Blind incontinent of urine 4 years. Weak	Cystitis Pernicious anemia	Suppurative nephritis with abscesses and pyonephrosis	Yes	Ascending urinary infection had preceded fracture
78	III	Buck's traction	40	Hemiplegia	Hypochromic, microcytic, anemia, Arteriosclerotic heart disease	Bronchopneumonia Cystitis	Yes	Reported transfusions corrected anemia but general condition was never good
91	III	Buck's traction	18	Tremor, orthopnea, haemorrhoids melena	Congestive failure	Acute dilatation of heart, Chronic hemorrhagic gastritis Senility	Yes	Patient stated he desired no treatment, only to die in peace
70	III	Buck's, Russell's and bilateral traction	79	Parkinson's disease Generalized arteriosclerosis	———	Confluent bronchopneumonia Acute and chronic myocarditis	Yes	Skin broke down in skin traction and required skeletal traction. Had ulcers on back, leg and foot - dermatitis made surgery hazardous. Fracture had healed
83	III	Buck's	30	Diabetes. Tabs frequently	Obesity Senile dementia Urinary and fecal incontinence	Bronchopneumonia	No	Gradually deteriorating course
94	III	Russell's traction	21	Senility	Senile dementia Incontinent urine and feces	Bronchopneumonia	No	Skinhead she was going to die
84	III	Russell's traction	23	In county nursing home Senility	Coma Dehydration Fractured right humerus	Generalized arteriosclerosis Terminal Bronchopneumonia	No	Was never in operable condition
89	III	Russell's traction	16	Senility - unable to determine prior history	Emaciated	Senility	No	Was never in operable condition
84	I	Russell's traction	25	Senility - unable to determine prior history	Emaciated Incontinent urine and feces	Pulmonary embolism Encephalomalacia Chronic focal encephalitis	Yes	Was never in operable condition

Analysis of time required and results obtained in the three groups is of interest, and although the numbers are small the differences are believed to be significant, and not explainable on the basis of the average age of patients sustaining Group III fractures being greater. See Table II.

TABLE II

COMPARISON OF TIME IN TRACTION AND ANATOMICAL RESULTS OF THE THREE GROUPS OF FRACTURES

Group	No. of Patients	Average Age	Average time in traction (days)	Average time solid union (days)	Cases were over 15°
I	10	75	35	51	0%
II	7	72	41	68	0%
III	19	78.5	45	88	6%

Complications During Treatment in Hospital

Bedsores, varying from reddened areas, which subsided without breakdown, to frank decubitus ulceration, were encountered in six of the nine nonsurviving patients and in 16 of the 36 survivors. These were all small, superficial, and were usually located over the sacrum. When breakdown occurred they were usually healed by the time the traction was discontinued. In no instance did the decubitus appear to be a factor in the mortality and none required surgical treatment. It was observed that they were apt to appear at about the end of the first week and to heal fairly rapidly as the patient became pain free and more cooperative regarding back care.

*All time intervals were from date of admission to hospital.

Urinary infection was noted in three of the non-surviving group and four of the survivors. In all of these cases it is believed to have antedated the fractured hip.

Respiratory infections other than frank pneumonia were noted in four of the surviving patients. Pneumonia occurred as a clinical diagnosis in eight of the non-surviving patients and four of the survivors. Among the latter group the response to the usual antibiotics was satisfactory. In the former group it is difficult to evaluate the importance of the pneumonia in causing the fatality. It is of interest that in the four patients coming to autopsy pneumonia was considered as having been important in causing death in only one.

Fecal impaction was noted in one of the non-surviving and five of the surviving group.

Dementia of a transient nature and not sufficient to interfere with treatment was noted in two of the non-surviving group and 14 of the survivors. Major dementia was noted in five of the former group and one of the latter, in which case it required changing the treatment from Russell's to well-leg traction with improvement in mental status following.

Results as to Function

We have been pleasantly surprised to find that the majority of these patients are living and getting about in a manner consistent with their general physical condition. See Table III. Most of the group answered their own letters, and there was no evidence to suggest that the period of mental aberration which several had exhibited in the hospital while under treatment for their fractures had left any permanent changes. One patient had become psychotic after her return home, and has remained in that way for more than three years, but had exhibited no psychosis while in the hospital and had, in fact, had a completely uncomplicated course and an excellent anatomical and functional result. Two patients reported difficulty with knee motion, one "poor" and one "fair" result (both hemiplegics) and one "good" result reported occasional difficulty with knee motion. This certainly does not suggest that this non-operative treatment was in itself detrimental to the knee in a permanent sense. No peroneal palsies were noted in this group.

Summary

A series of 45 trochanteric fractures treated non-operatively during the past five years has been reviewed. It is a selected series in that it includes those patients who were not in operative condition on one hand and those who looked as if they would do as well, or better, in traction than with operative fixation on the other. The average age of these patients was 77.6 years; the mortality rate was 20 per cent; the average age of the survivors was 76 years, of the non-surviving group 84.1 years. Twenty-five of the 36 survivors are known to have become ambulatory again, six did not because of conditions not related to the fracture or the treatment and five could not be traced sufficiently to determine the result accurately. The anatomical results of treatment in traction were generally good in Group I and II fractures, and in many Group III fractures. However, six of 19 of these latter united with a coxa vara of 115 degrees or less. No patient with this much coxa vara is known to have had an excellent or good result, three of them had fair results, and one had a poor result. The treatment and complications have been described.

TABLE III

FOLLOW-UP DATA ON 36 SURVIVING PATIENTS

Results	No.	Average Age	Average Length Follow-up	Group I	Group II	Group III	Coxa vara of 115 or less
Excellent	8	71.7	36 mos.	5	1	2	0
Good	8	73.3	38 mos.	2	1	5	0
Fair	6	78	36 mos.	1	1	4	4
Poor	3	81	34 mos.	0	0	3	0
Satisfactory fracture healing but disabled completely by other disabilities	6	74	22 mos.	1	3	2	0
Insufficient data to grade results	5	80		1	1	3	3
				10	7	19	7

Conclusions

Russell's traction remains a useful method of treatment in selected patients with trochanteric fractures of the femur. It seems probable that improved medical management including antibiotics has rendered it a less hazardous method than it was a decade ago.

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The Effects of ACTH on Liver Function*

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The experimental background of adrenal steroid physiology lends ample support to the contention that intimate relationships exist between the adrenal cortex and liver function. Experimental adrenalectomy produces a hypoglycemia[1] and loss of glycogen[2] from the liver of experimental animals. By the use of adrenal cortical extract this defect can be corrected with a restoration of the liver glycogen.[3] The liver glycogen reaches a maximum with increasing doses of extract while elevation in the blood sugar and glycosuria continue to increase. The increase[4] in the liver glycogen is accompanied by an increased excretion of nitrogen suggesting that protein is being converted to carbohydrate while studies on liver slices[5] have shown a marked increase in the production of glycogen from pyruvate and d-lactate under the influence of adrenal cortical hormone. These effects of the adrenal cortical extract have been produced by 17-hydroxy-11-dehydrocorticosterone but not by desoxy-corticosterone acetate.[6]

In partially depancreatized animals the production of the glycosuria with adrenal cortical extract is associated with a ketonuria and acidosis, again pointing to the liver cell as the effector for the adrenal cortical extract, since the production of ketone bodies is known to depend on an intact liver.[7]

ACTH causes an increase in the fat vacuoles[8] in liver of rats fed on a high carbohydrate diet during the treatment period but not in animals fed on a high protein diet. Some disagreement exists as to whether the "fat metabolizing factor" of pituitary extracts is actually ACTH,[9] but it has been shown that this deposition of fat in the liver by pituitary extracts can be prevented by adrenalectomy.[10]

Cortical hormones cause an increase[11] in the serum albumin and a decrease in the serum globulin which again implicates the liver in this effect since it is known to be instrumental in the production of the serum proteins.

Some of the cellular enzymes of the liver have been shown to be influenced by steroid hormones. The alkaline phosphatase of the liver is increased by steroids with oxygen in the C-11 position but the arginase is not altered.[12] Conversely, the liver in vitro has the ability to alter chemically the steroid hormones,[13] and to "inactivate" them.

Berman, Sylvester, Hay, and Selye[14] reported that in experimental animals adrenalectomy decreased the deposition of fat, inhibited the deposition of protein and prevented the increase in size and number of regenerating cells following partial hepatectomy. Adrenal extract corrected for these changes while DOCA did not. Rapport, Canzanelli and Guild[15] also demonstrated a reduction in regeneration of liver cells following adrenalectomy. Desoxyribosenucleic acid was increased following adrenalectomy. Freidgood, Vars, and Zerbe[16] noted that adrenalectomy impaired the deposition of protein in the liver following partial hepatectomy and that DOCA restored the protein synthesizing power of the liver. The regenerative power of the liver was restored by cortical extract.

These are some of the experimental data pointing to the liver as an important target organ for the effect of adrenal cortical hormones. Changes in liver function as evidenced clinically and in the laboratory might be expected to occur under the influence of adrenal cortical hormones; however, there are as yet only a few references in the literature to these alterations. Bluemle[17] et al reported three cases of chronic liver disease treated with ACTH with apparently an acceleration in the clinical improvement. Bongiovanni[18] et al reported seven patients with cirrhosis of the liver treated with ACTH with little noticeable clinical effect but significant improvement in the serum albumin values. They mentioned bloody ascites and elevation of the blood sugar as undesirable side effects occuring in each of two patients.[19] Webster reported nine patients with acute and chronic liver disease treated with adrenal cortical extract with very promising clinical results although the course of treatment with cortical extract was for only a short period.

In the light of such experimental data, adrenal cortical treatment in liver disease might be expected to produce either beneficial or detrimental effects. The increase in the liver glycogen should serve to protect the organ against further damage and the supressive effect on tissue inflammatory reaction exerted by cortical compounds might mitigate the development of fibrotic changes. The effect on regeneration of liver cells should also be beneficial. On the other hand the role of the adrenal in the production of fatty changes in the liver poses, at least superficially, a disturbing question, since such changes are a prominent feature of the histological changes in liver disease.

Material

The patients in this group were divided into two groups.

*From the Department of Internal Medicine, University of Kansas School of Medicine.

Group I. Twenty-one patients undergoing treatment with ACTH for various conditions, but showing no evidence clinically or biochemically of liver disease, were studied with liver function tests before treatment and during or immediately after treatment.

Group II. This consisted of 11 patients showing evidence of liver dysfunction, five of which showed clinical liver disease and seven of which showed only elevated cephalin cholesterol flocculation test in the presence of other disease processes. Liver function tests were done before treatment and late in the course of treatment.

The treatment consisted of injections of ACTH at six hourly intervals totaling from 40 to 60 milligrams per day for a period of approximately two weeks. One additional patient not included in either of the above groups received adrenal cortical extract. Other adjuncts to treatment are listed in the case presentations below. Liver punch biopsies were done before treatment on all of the cases showing clinical hepatic disease and on three of the cases after treatment.

Case Reports

Case 31, E. A. Diagnosis: Hanot's cirrhosis. This 43-year-old white male was first admitted with the complaint of jaundice for two or three weeks. The illness had begun four or five months before admission with feeling of weakness and malaise and progressively increasing anorexia, and occasional spells of vomiting after eating. A definite history of alcohol was obtained.

Physical examination revealed moderate jaundice. The tongue was a beefy red. The abdomen was quite distended with ascitic fluid and the liver edge was palpable five centimeters below the costal margin and non-tender. Spider angiomata were noted about the upper part of the chest and shoulders.

Red blood count was 3,590,000 with nine grams of hemoglobin, sugar 110 milligrams per cent, blood chloride 94 meq., carbon dioxide combining power 20.6 meq., cholesterol 300 milligrams per cent with 40 per cent esters, bromsulphthalein (two milligrams at 20 minutes) 15 per cent retention, total cholesterol 300, prothrombin time 70 per cent of normal, urinary urobilinogen three, fecal urobilinogen 64 milligrams.

Punch biopsy of the liver showed fatty metamorphosis and acute and chronic hepatitis with diffuse fibrosis.

Course in hospital: The patient was given ACTH, 40 milligrams per day for 17 days, and demonstrated an immediate increase in appetite to the extent that he was able to take a full Patek type of diet, and showed marked reduction in his nervous irritability. The beefy red tongue returned to a normal color, the spider nevi disappeared, and the liver became reduced in size by about three centimeters. Urinary output increased from an average of 600 cc. a day to 4,000 cc. per day. The patient was seen four months later and had demonstrated continued improvement with a weight gain of 40 pounds without evidence of fluid retention.

Discussion: This case demonstrates the rapid improvement from a state bordering on complete hepatic failure as evidenced by the deepening jaundice, fluid retention and increased nervous irritability. The reversal of these trends along with a rise in the hemoglobin was definitely evident three days after the institution of ACTH therapy. It is difficult to say whether a primary effect of the ACTH was responsible for this improvement or whether it stemmed secondarily from the remarkable improvement in the patient's appetite with ability to consume a high protein diet.

Case 32, K. S. Diagnosis: Laennec's cirrhosis with bleeding esophageal varices. This 49-year-old white female was first admitted to the Medical Service November 30, 1949, with the diagnosis of Laennec's cirrhosis. In January of 1950 she was discharged from the hospital with a Patek diet and had done fairly well until one month before the present admission on August 13, 1950. Prior to this admission she had been having increased weakness, exertional dyspnea, a reappearance of ascites, anorexia, and hematemesis. A mild icterus was noted, and spider nevi were present on the face and left arm. The heart was slightly enlarged. The abdomen was markedly distended with ascites and was too turgid to allow palpation of the liver. The lower extremities were edematous.

Admission blood count showed 3,250,000 red cells, 9.5 grams hemoglobin. Sugar was 74 milligrams per cent, serum chloride 98.5 meq.; carbon dioxide combining power 23.2 meq.; B.S.P. five per cent retention; serum bilirubin 2.5 milligrams; alkaline phosphatase 24 units; cephalin cholesterol, one plus; thymol turbidity, 12 units; serum albumin one gram; serum globulin 3.5 grams; total cholesterol 200 with 40 per cent esters; prothrombin 40 per cent of normal, with no rise after vitamin K. Glucose tolerance test showed fasting blood sugar 174, first hour 279, second hour 304, third hour 270, fourth hour 325, fifth hour 293. A liver biopsy was done on the first admission and showed periportal fibrosis with some variation in the nuclei of the liver cells.

Course: The patient's urinary output was only 400 cc. a day. It rose to 1800 cc. on the eighth hospital day and again fell on the 12th hospital day. The patient had episodes of vomiting of coffee ground material while in the hospital which continued throughout her stay, and it was concluded that she

was bleeding from esophageal varices. ACTH treatment was started on the eighth hospital day following which there was a marked improvement in the patient's appetite and feeling of well being. Evidence of bleeding continued, however, and became more pronounced. On her 12th hospital day she was given 50 milligrams of benadryl and shortly thereafter developed a comatose state which, however, disappeared after about 24 hours. A serious exacerbation of her bleeding occurred on the 15th hospital day and in spite of transfusions and supportive therapy the patient expired.

Discussion: This patient showed a far advanced Laennec's cirrhosis complicated by persistently bleeding esophageal varices and a decreasing hemoglobin. ACTH therapy appeared to exert no influence on the course of the fundamental process although it did produce a salutary effect on the patient's appetite and feeling of well being. One may speculate that if such treatment had been instituted earlier in the course of this patient's disease it might have helped to reserve the vicious cycle of malnutrition and further liver damage.

Case 33, J. W. Diagnosis: Hemochromatosis, cirrhosis, delirium tremens. This 58-year-old white male was admitted with the complaint of bloating and constipation for two weeks, varicose ulcers on the legs and chest pain for two months. A strong history of alcohol was obtained. The patient was disoriented and confused, very tremulous and uncooperative. The tongue was unusually red. The liver was palpable in the midline eight centimeters below the costal margin and was slightly nodular and non tender. Edema or ascites were not noted.

Essential laboratory work showed a blood sugar of 95 milligrams per cent, blood chloride, 89 meq., carbon dioxide combining power 13.5 meq., cholesterol 210 milligrams per cent with 56 per cent esters; prothrombin time 80 per cent of normal. Punch biopsy of the liver showed large amounts of pigment in the portal spaces and liver cells, which showed a positive test for iron, and wide bands of proliferated fibrous tissue containing small round cells and macrophages transversing the lobules. Numerous fat deposits were noted. The pathological diagnosis was cirrhosis of the liver compatible with hemochromatosis.

Course: The patient was treated with 40 milligrams a day of ACTH for nine days. At the end of the first day of treatment he showed marked subsidence in the maniacal symptoms. Prior to treatment, the temperature had risen to 104 degrees F. and immediately subsided after treatment was started. The patient developed a voracious appetite after treatment was started which persisted throughout his hospital stay. The liver decreased in size by about two centimeters during treatment. Hepatic function test showed little change, except for an elevation of the cephalin cholesterol to two plus during the course of treatment and a return to zero after termination.

Discussion: The two factors which were largely responsible for the clinical picture on this patient were the alcoholic delirium tremens state superimposed upon chronic moderately advanced liver disease. The most notable effect following the institution of ACTH treatment was in the acute manifestations of the delirium tremens with a prompt subsidence in the maniacal symptoms and of the fever with a return of the appetite, such that a high protein intake was again feasible. Changes in the underlying chronic liver disease could be considered only equivocal as seen in the slight but definite decrease in the size of the liver. The acidosis was corrected; however, this cannot be ascribed directly to the ACTH since the patient was given additional fluid therapy.

Case 34, N. H. Diagnosis: Laennec's cirrhosis. This 64-year-old white male was admitted complaining of diarrhea and bloating with indigestion for about six to seven weeks. He had noted a diminution in his urinary output. An alcoholic history was obtained. The heart was enlarged and the chest somewhat emphysematous. The abdomen was markedly distended with fluid and the liver could not be palpated because of the turgidity of the abdomen.

Admission red cell count was 3,700,000 with 10 grams hemoglobin. Wassermann and Kahn were four plus. Sugar was 75 milligrams per cent; cholesterol was 178 milligrams with 58 per cent esters.

A liver biopsy was attempted on two occasions, but because of the marked ascites and small fibrous liver it was not highly successful, showing only a small amount of liver tissue. The patient was given ACTH for 10 days at 40 milligrams per day. In addition to this, the patient was given mercurial diuretics and a high protein diet. During the treatment the patient became depressed, developed crying spells and showed a progression in the fluid retention with a weight gain of 12 pounds. At the conclusion of treatment, the urinary output increased and the patient lost the accumulated fluid until his weight was the same as initially. After being dismissed from this hospital, the patient was hospitalized elsewhere and laparotomy performed. The diagnosis of Laennec's cirrhosis of the liver was confirmed and the hepatic artery tied off at operation. The patient expired several days after operation.

Discussion: Again, this is a patient showing far advanced Laennec's cirrhosis with marked fluid retention. ACTH seemed to exaggerate the tendency to fluid retention and in addition produce an un-

favorable psychic response, both of which disappeared, however, at the termination of treatment. No beneficial effect can be ascribed to ACTH.

Case 35, R. M. Diagnosis: Laennec's cirrhosis. This 39-year-old white male was admitted with the chief complaint of anorexia and diarrhea of two weeks duration. There had been a 20 pound weight loss during the past six months. He had been a chronic alcoholic for 10 years. The patient was moderately jaundiced and large spider nevus was seen on the forehead. The chest was moderately emphysematous. The liver was five centimeters below the right costal margin and the spleen was palpable one centimeter below the left costal margin.

Blood count was 2,220,000 red cells, 7.4 grams hemoglobin, 38,000 white cells with 87 per cent polymorphonuclears, five per cent lymphocytes, six per cent monocytes and two per cent metamyelocytes. Blood sugar was 80 milligrams per cent; blood chloride, 90 meq.; carbon dioxide combining power, 21.8 meq.; cholesterol 148 milligrams per cent, 29 per cent esters; BSP, 10 per cent retention at 20 minutes. Liver biopsy showed hepatic cell cytoplasm to be granular and vacuolated, often completely replaced. Considerable hepatic cell nuclear variation and increase in fibrosis was noted. Pathological diagnosis was fatty metamorphosis, fibrosis of the liver, and acute and chronic hepatitis.

Course: During the first few days of the patient's hospitalization he became slightly disoriented and somnolent. He was then given a course of ACTH, 40 milligrams a day for eight days and 60 milligrams a day for four days and 30 milligrams a day for three days. After four days of treatment an improvement in the appetite occurred along with an improvement in the patient's sensorium. Improvement lasted for several days and then while the patient was on treatment there was a recurrence of his symptoms. He left the hospital with no change in status from his condition on admission. Contact was lost with the patient as he returned to his home community; however, it was later learned that he had died. No autopsy was obtained.

Discussion: A definite but brief salutary effect was noted during ACTH treatment but the relapse occurred during treatment. This would suggest that ACTH probably did not affect the progress of the fundamental process of liver disease, but probably exerted some effect on the manifestations of the disease, notably the anorexia, nervous irritability and somnolence.

Case 47, G. P. Diagnosis: Laennec's cirrhosis. This 59-year-old white male was admitted complaining of hemorrhoids of two years duration. He was a chronic alcoholic and the liver disease was found incidentally. The odor of alcohol was evident on the patient's breath. The abdomen showed the liver to be four centimeters below the right costa margin, firm, tender, and slightly nodular. Ques tionable fluid was present. Large hemorrhoids were noted, both internally and externally.

Blood sugar was 95 milligrams per cent; bloo chloride, 93 meq.; carbon dioxide combining powe 20.3 meq.; serum cholesterol 318 milligrams pe cent with 69 per cent esters; bromsulfalein, five pe cent retention.

Liver puncture biopsy before treatment showe wide bands of proliferated fibrous tissue focally an dense infiltration with leukocytes, chiefly lympho cytes and proliferating bile ducts. Liver cells neares these areas contained large multiple nuclei.

Course: The patient was treated with ACTH, 4 milligrams a day for 14 days (the ACTH was increased to 60 milligrams a day for five days during the middle of the treatment course).

Discussion: There seemed to be neither a beneficial nor detrimental effect in any way on this patient. It is noted that at the time of admission he was not suffering from any acute manifestations of liver disease, which was picked up only incidentally in the course of examination for hemorrhoids, which may have stemmed originally from the cirrhosis.

Case 52, F. R. Diagnosis: Biliary cirrhosis, chronic biliary obstruction. This 44-year-old white female had had jaundice following cholecystectomy for 16 years which was unrelieved by numerous surgical procedures. She was admitted to this hospital August 18, 1950, and further surgery was performed to relieve the biliary obstruction which resulted in a biliary fistula. The patient was emaciated and showed a generalized pigmentation resulting from the chronic jaundice.

Liver function tests on admission showed alkaline

TABLE I. Effect on Hepatic Function Tests

Case	Diagnosis	Alk Phos			Hanger's			Th Turb			Alb/Glob		
		B	A	C	B	A	C	B	A	C	B	A	C
Group I													
2	Rheum Arth	0	0	0	0	0	0	5	1	-4	1.1	1.6	0.5
3	Rheum Arth	1.3	9.0	8.2	0	0	0	4	6	2	2.9	2.3	0.3
5	Rheum Arth	6.3	9.4	3.1	0	0	0	7	3	-4	1.6	1.5	-0.1
7	Rheum Arth	9.9	0.1	-9.8	0	0	0	5	1	-4	1.4	2.0	0.6
9	Rheum Arth	4.0	4.1	0.1	0	0	0	4	6	2	1.8	2.0	0.2
11	Rheum Arth	0.5	8.0	7.5	0	2	2	5	5	0	1.6	1.6	0
14	Emphysema	6.9	5.0	-1.9	0	0	0				1.0	1.2	0.2
15	Emphysema	10.0	7.5	-2.5	1	0	-1	3	2	-1	1.8	1.2	-0.6
16	Asthma	5.6	5.2	-0.4	0	0	0	3	5	2	2.1	2.0	-0.1
20	Dermatitis	2.0	6.8	4.8	0	0	0	1	2	1	2.0	1.6	-0.4
21	Retinitis	8.1	5.3	-2.8	0	0	0	5	7	-5	2.5	2.6	0.1
22	Retinitis	4.9	1.7	-3.2	0	0	0	5	7	2			
23	Choroiditis	7.5	6.6	-0.9	0	0	0	5	5	0	1.6	1.6	0
26	Purpura	10.0	10.0	0	0	0	0	5	4	1	2.5	2.2	-0.3
41	Xanthoma	5.0	6.2	1.2	0	0	0	4	4	0	1.5	1.8	0.3
42	Rheum Arth	5.2	1.8	-3.4	0	0	0	3	4	1	1.8	2.2	0.4
43	Rheum Arth	10.0	5.2	-4.8	0	0	0	3	2	-1	0.8	0.8	0
44	Retinitis	2.7	2.7	0	0	0	0	3	2	-1	1.5	1.6	0.1
46	Rheum Arth	6.8	4.0	-2.8	1	0	-1	4	5	1	1.8	2.2	0.4
50	Retinitis	7.0	2.0	-5.0	0	0	0	3	1	-2	1.6	2.0	0.4
51	Ulcer. Colitis	18.0	15.0	-3.0	0	0	0	3	4	1	0.4	0.7	0.3
Group II													
4	Rheum Arth	6.7	9.1	2.4	4	4	0	8	10	2	1.7	1.7	0
8	Rheum Arth	12.0	6.2	-6.8	3	0	-3	7	4	-3	1.1	2.0	0.9
10	Rheum Arth	6.9	7.0	0.1	2	3	1	8	5	-3	1.8	1.0	-0.8
18	Asthma	7.0	9.0	2.0	2	2	0	2	2	0	0.8	1.8	1.0
31	Cirrhosis	19.0	11.0	-8.0	3	2	-1	22	5	-17	0.5	1.1	0.6
33	Cirrhosis	10.0	5.6	-4.4	0	2	2	2	4	2	1.7	1.8	0.1
34	Cirrhosis	15.0	16.0	1.0	3	4	1	24	18	-6	0.8	0.7	-0.1
35	Cirrhosis	24.0	37.0	13.0	4	1	-3	0	2	2	1.0	0.8	-0.2
45	Rheum Arth	5.5	9.0	3.5	3	4	1	7	6	-1	0.7	1.4	0.7
47	Cirrhosis	11.0	7.5	-3.5	3	1	-2	5	4	-1	1.6	1.1	-0.5
38	Multiple Scler.	8.4	11.0	2.6	2	2	0	3	4	1	1.5	1.5	-0.1

Abbreviations: B,Before treatment; A,End of treatment; C,Units of change during treatment; Alk Phos, Alkaline Phosphatase (King-Armstrong);Th Turb,Thymol Turbidity; Alb/Glob, Albumin-globulin ratio;Rheum Arth, Rheumatoid arthritis.

phosphatase, 34 units; serum bilirubin, 12.0 milligrams per cent; chephalin cholesterol, four plus; thymol turbidity, six units; serum albumin, 2.0; serum globulin, 4.0; total cholesterol, 250 milligrams with 36 per cent esters; and prothrombin time 20 per cent of normal.

She was followed with supportive treatment and on November 3 exhibited a drop in blood pressure and gradually became more weak and irrational. The serum sodium was 118 meq. and the serum potassium 7.9 meq., and the N.P.N. 76 milligrams per cent.

Therapy was started with adrenal cortical extract (aqueous) with a rapid return to the rational state within 24 hours and improvement in well being. The extract (Upjohn) was given in five cc. amounts twice daily. After 14 days of treatment the serum electrolytes were sodium 139 and potassium 5.4 meq. The patient expired on November 27, 1950.

Discussion: In the course of chronic biliary obstruction this patient developed a picture resembling acute adrenal insufficiency with characteristic clinical and electrolyte changes. This syndrome responded to treatment with cortical extract with a prolongation of life for 14 days (Liver function and glucose tolerance data are shown in Table I).

Histological Observations

Case 31, E. A. (Figures 1 and 2). The section taken before treatment shows wide bands of dense collagenous tissue with scattered areas of bile duct proliferation. The liver cells themselves are shrunken and mis-shapen. Vacuolization of the cytoplasm is extensive with many areas showing hardly any identifiable liver cells. The section, obtained after treatment, is from an area which possesses much less fibrosis than the pre-treatment slide, making it difficult to interpret any change in the stroma. A striking difference is noted in the liver cells in that they are larger, showing the normal polygonal shape and having lost the shrunken appearance noted in the previous section. The cytoplasm has a foamy reticulated appearance and vacuoles are quite rare. Some sections were stained with Gomori's[20] silver stain for glycogen and there is seen a marked increase in the glycogen in the cell following treatment. The amount of glycogen in the cell is great enough that it could account for the increase in the size of the liver cell as well as the reticulated foamy appearance of the cytoplasm noted on the routine stains. Sections stained with Schiff's reagent for the Feulgen reaction reveal an increase in the size of the nucleus and an apparent decrease in the concentration of desoxyribose nucleic acid, with a more distinct nucleolus on hematoxylin stain. Stains for alkaline phosphatase reveal no change from the pre-treatment picture. Likewise stain for esterase shows no detectable change.

Case 33, J. W. (Figures 3 and 4). The section taken before treatment shows the liver cells to be small and shrunken with many areas of extensive vacuolization of the cytoplasm. The nuclei are dark staining and pyknotic. The iron pigment of hemochromatosis is present. Following treatment the section shows the cytoplasm to be reticulated and foamy while the areas of vacuolization have largely disappeared. The nuclei are much larger and lighter staining and the nucleoli much more distinct. Silver stains reveal a noticeable increase in glycogen. No change is noted in the sections stained for alkaline phosphatase and esterase.

Case 47, G. P. The sections taken before treatment do not show any definite abnormality in the liver cells and the sections taken after treatment likewise reveal no change.

Discussion. The most noticeable changes are in the liver cells as shown in cases 31 and 33 where changes occur both in the cytoplasm and nucleus. The cytoplasmic changes seem to consist mainly of

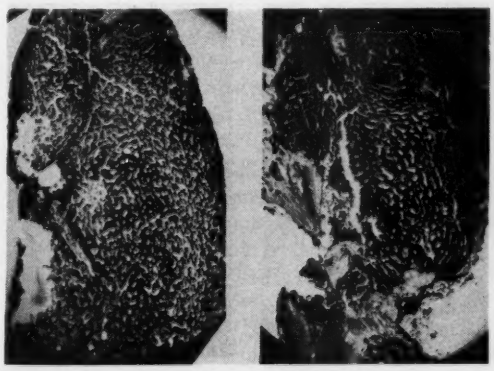

Figure 1. Case 31, E. A. Liver punch biopsy stained for glycogen by the method of Gomori. The section on the left is taken before treatment and the one on the right after treatment (X 100).

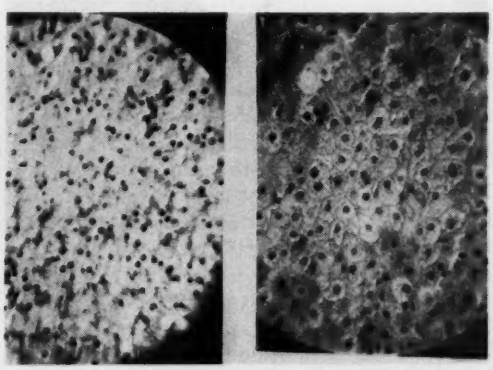

Figure 2. Case 31. E. A. Liver punch biopsy stained by routine H and E. The section on the left is taken before treatment and the one on the right after treatment (X 440).

a loss of fat vacuoles and an increase in the glycogen in agreement with similar changes reported in experimental animals under the influence of cortical extracts. The disappearance of the fat vacuoles is not entirely in agreement with the animal experiments, but it has been shown[8] that the extent of the fat deposition in the liver depends largely on the diet given to the animals at the time of administration of cortical extract. When a high carbohydrate diet is given the liver cells are more likely to show the vacuolar changes, while if a high protein diet is given they are less likely to do so. The condition in these patients parallels more closely those experiments in which a high protein diet was given since the Patek diet was administered. Such an alteration in the cytoplasm of the cell should conceivably be associated with changes in the intracellular enzymes; however, no changes are noted in these sections. Such changes as have been described in animals[12] might have followed had the treatment period been longer. Case 47 which showed no abnormality in the liver cell before or after treatment was clinically a compensated cirrhosis which was discovered incidentally.

Effect on Liver Function Tests

The results of the liver function tests are summarized in Table I. In neither group is there any statistically significant change in the alkaline phosphatase, although variations in either direction are common. No significant change is noted in the serum bilirubin, thymol turbidity or the bromsulfalein excretion.

Only three of the patients in Group II show a decrease in the cephalin cholesterol flocculation from pre-treatment values while the rest show slight increases. Three patients treated with cortisone show increases in the flocculation. No consistent change is noted in the serum albumin. In 13 of 19 patients in Group I there is a small decrease in the serum globulin while a smaller number of patients in Group II show a decrease. These findings[11] are generally consistent with other reports in the literature to the effect that globulin decreases under adrenocortical hormone treatment. In Group I, 12 of 19 patients show an increase in the albumin-globulin ratio which is mostly due to the change in the globulin fraction, while a smaller number in Group II show this change. Since the changes in the serum proteins occur quite as commonly among the patients with no evidence of liver disease, it is doubtful that these determinations or any of the determinations depending upon the protein structure of the plasma have very much validity as a reflection of liver function during ACTH or cortisone therapy.

The glucose tolerance tests are shown in Table II.

TABLE II. Effect of ACTH on Glucose Tolerance Test

Group I

Case	Before Treatment					After Treatment					Change in 2nd hour specimen
	F	1	2	3	4	F	1	2	3	4	
6	78	200	265	225	160	95	214	270	248	169	5
7	103	178	214	197	155	107	190	200	187	128	-14
14	75	160	123	67	50	103	132	178	140	85	55
15	85	90	169	80	50	85	225	187	150	89	18
16	73	155	150	110	120	67	100	89	75	59	-61
20	83	160	126	92	112	150	170	165	96	106	35
21	97	140	108	54	70	72	200	140	50	63	132
22	75	120	100	92	95	70	107	103	92	85	3
23	103	245	135	107	92	100	245	204	169	144	69
41	92	135	92	120	60	75	190	169	110	89	77
42	95	135	95	128	63	89	128	122	63	70	-27
43	145	245	315	335	365	128	209	237	248	320	-78
46	103	214	174	135	85	80	107	54	50	60	-120
51	54	165	200	160	80	120	256	206	100	112	6
								Mean difference			10

Group II

4	71	100	150	169	85	76	107	132	95	75	-18
31*	107	209	190	103	85	100	165	169	132	56	-21
33*	95	182	169	123	120	92	200	204	182	209	35
34*	75	230	204	200	160	258	365	382	408	358	178
35*	85	165	118	80	76	120	135	128	118	100	10
45	85	209	110	160	-	89	182	170	95	67	60
47*	92	174	110	56	63	92	95	-	67	67	-
								Mean difference			40

* Cases of Clinical Liver Disease.
The last column indicates in milligrams per 100 cc, the change in the second hour specimen after treatment compared to the second hour specimen for the pre-treatment glucose tolerance test.
Difference of the means 30
Standard Error ±10
P is <.01

The Group I patients show a mean increase in the second hour specimen of 10 mg. while the Group II patients show a mean increase of 30 mg. This difference is statistically significant and does show a definite trend toward exaggeration of the diabetic state in cases of liver disease. This does not seem

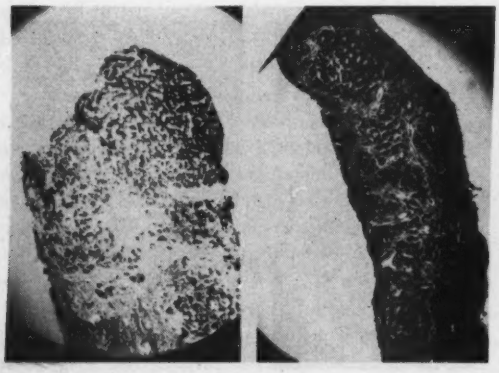

Figure 3. Case 33, J. W. Liver punch biopsy stained for glycogen. The section on the left is taken before treatment and the one on the right after treatment (X 100).

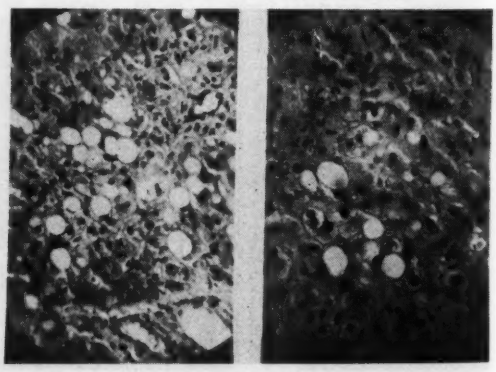

Figure 4. Case 33, J. W. Liver punch biopsy stained by routine H and E. The section on the left is taken before treatment and the one on the right after treatment (X 440).

to be a permanent defect, however, as it clears up after treatment. Two of the cases of liver disease which showed an increased diabetic tendency during treatment actually show more normal curves after treatment than before, associated in each case with clinical improvement in the liver disease.

Two factors are operating to decrease the glucose tolerance during ACTH therapy: one is the increased conversion of protein to carbohydrate and the other is decreased utilization of glucose in the tissues. The added factor of liver disease with its tendency to lowered glucose tolerance[21] may augment the diabetogenic effect of ACTH in some way. The decreased glucose tolerance attending ACTH therapy differs from ordinary diabetes in several respects: an increased insulin resistance has been reported, and there is little tendency to acidosis, even though corticoids have been shown to be active in the formation of ketone bodies. The tendency of the corticoids to cause alkalosis by effects on electrolyte excretion perhaps may oppose the production of acidosis. Another difference is noted in the histological sections since characteristically the liver in unregulated diabetes is depleted of glycogen while the liver in such a case of corticogenic diabetes shows increased glycogen.

Discussion

The data presented support the idea that the liver forms a significant link in the chain of reactions resulting from adrenal cortical stimulation in the human. Two questions might be considered regarding the role of the liver in this reaction: do adrenal cortical hormones alter liver function in normal or disease states and does liver dysfunction alter the effects of adrenal cortical hormones in the body?

The improvement in the clinical condition of patients with liver disease, the increased glycogen noted histologically following treatment with ACTH, and decrease in the number of fat vacuoles in the liver cells lend support to an affirmative answer to the first question. In the liver function tests themselves no particular change is noted except in the serum globulin, a change which cannot be ascribed directly to changes in liver function but which is likely to affect the liver function tests to some extent.

An answer to the second question is not immediately forthcoming, although in this work there is a tendency for the diabetogenic action of ACTH to be exaggerated in the group showing liver disease. The fluid retaining effects of the treatment are more noted in the cases with liver disease where fluid retention has been present before. One might speculate that other responses to the adrenal cortical hormones are also conditioned by the status of liver function by analogy to other steroids such as estrogen[22, 23] where the evidence is strong that liver dysfunction alters the response. In these cases the liver is not only able to conjugate the steroids but can effect changes in the substituting groups[13, 24] attached to the ring, and since the functional capacity of any steroid is highly dependent on the structure of the substituting group, any alteration in this mechanism might be expected to give rise to changes in the response to steroid. Work along this line is needed particularly in the field of intermediary metabolism of the 11-oxygenated steroids of the adrenal cortex.

The mechanism whereby the adrenal cortical hormones affect the liver remains a question for which only partial answers exist. The changes in carbohydrate metabolism are at present the most striking and therefore point to this as the primary mechanism. The storage of glycogen in the liver is evident in these studies, directly on the histological sections, and indirectly by the decreased glucose tolerance. The decrease in glucose tolerance could arise by several mechanisms, one of which might be the saturation of the liver cells with glycogen and a consequent slowing of glycogenesis, due simply to the mass action effect of the glycogen, as well as the decrease in utilization of glucose in the peripheral tissues.

The changes in carbohydrate metabolism are probably secondary to changes in the cellular enzymes, although these studies did not show any evidence of tissue esterase or alkaline phophatase increase, but are not conclusive. Whether the changes in fat metabolism are secondary to the changes in carbohydrate metabolism or are a primary effect of the adrenal hormone cannot be answered. The disappearance of fat vacuoles from the liver cells in the two observed cases was definite and occurred along with the increase in glycogen. This could follow from decreased delivery of fat to the liver or increased utilization of fat from the liver, by the tissues.

The enlargement of the nuclei of the liver cells points strongly to an improved functional state of the liver cell, although definite evidence of increased cell division is not obtained.

The clinical improvement found in several of these cases was definite but seemed to be of a nonspecific nature, and similar to the improvement seen in patients with other types of disease when treatment with adrenal hormones was used. These changes consisted of increased appetite, increased feeling of well being, followed by a gradual decrease in the signs of liver disease such as the hepatomegaly, spider nevi, and discoloration of the tongue. In one case where impending hepatic coma was evident there was a marked diminution in the symptoms with a continued improvement in the condition of the patient.

Any improvement in the signs of liver disease followed the appearance of these non-specific changes, so the most logical explanation for this is that the medication gives rise to a good appetite which allows the patient to consume large quantities of protein and thereby allows the liver disease to heal. Such a situation may be the factor that is necessary to alter the course in the chronic disease process from one of gradual deterioration to one of continued improvement. The appearance of a syndrome not unlike adrenal cortical insufficiency and corrected by adrenal cortical extract is suggestive that actual insufficiency may appear in the course of liver disease.

Such a possibility is supported by the finding that the urinary 17-ketosteroids are decreased in liver disease.[25] The adrenal insufficiency might arise through some abnormal metabolism of the steroids in the diseased liver or by a deficiency of epinephrine-like substances[26] produced in the liver and a consequent lack of stimulation of the pituitary adrenal mechanism.

Summary

1. The literature is reviewed showing relationships between the adrenal cortex and hepatic function in the fields of carbohydrate and fat metabolism.

2. Case summaries are presented on seven patients, six of whom received ACTH and one of whom received adrenal cortical extract. Two of these patients showed sustained improvement, three showed temporary improvement, one showed no change, and one became worse. Two died during the course of treatment and two others died some time after leaving the hospital. It is doubtful that the treatment was instrumental in the death.

3. Histological changes demonstrated in two patients include increase in the size of the nucleus of the liver cell, increased reticulation of the cytoplasm, diminution of fat vacuoles, and increase in the amount of glycogen in the liver cells.

4. No significant changes are noted in the liver function tests. There is a tendency for the diabetogenic action of ACTH to be exaggerated in patients with liver disease.

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Reversible Cerebral Atrophy

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Gerard and Grinker[1] state that the view has been universally adopted that regenerative processes in the central nervous system of the higher vertebrates do not occur, though experimental work on animal embryos throws some doubt on this interpretation. Lichtenstein[2] states that regeneration of nerve fibers in the central nervous system of man does not occur. He states "loss of nerve cells is irreplaceable from an anatomic viewpoint. Physiological readjustment and recovery of partially damaged elements, however, result in clinical improvement. Regeneration of structures within the nerve cell, as for example, the Nissl substance, occurs if the vitality of the cell has not been too severely affected."

It is generally assumed that gross atrophy of the brain as shown on pneumoencephalogram, whether congenital, post-traumatic, post-encephalitic, arteriosclerotic, or due to unknown cause, is irreversible. Davidoff and Epstein,[3] in their monumental monograph on the abnormal pneumoencephalogram, make no mention of the possibility of gross atrophy of the brain, as verified by pneumoencephalogram, being reversible.

Because of the unusual clinical recovery, and the striking improvement in cerebral atrophy as shown by pneumoencephalography, the following case seems worthy of report.

Report of Case

J.M., a 3-year, 10-month old colored male, was admitted to the University of Kansas Medical Center October 5, 1948. He had become ill approximately three weeks previously with malaise, fever, vomiting, and diarrhea. Frequent generalized convulsions began three days after onset of illness, and after a week the child became lethargic and refused to talk. Within two weeks the patient had great difficulty in swallowing, ceased normal conjugate movement of the eyes, and lost the use of all extremities.

Examination revealed an afebrile child who lay immobile in bed except for an occasional slight movement of the eyes or head. He was completely conscious, yet seemed to stare straight ahead into space and would not talk. He lay with the legs in a frog-like position, arms extended, and fists clenched. There appeared to be almost total quadriplegia, though slight movements would occur on painful stimulation. The arms exhibited rigidity, yet the legs seemed flaccid. The deep tendon reflexes were present and equal bilaterally and were within normal limits. Babinski and Hoffman reflexes were negative. The child was unable to swallow or speak, and the gag reflex was decreased. The pupils were equal and reacted to light, and the eye grounds were normal.

The white blood count was 17,300 with 71 per cent polys, red cell count 4,230,000, urinalysis was negative, and blood calcium 11.2 mg. per cent. Analysis of the spinal fluid revealed no cells, sugar 47.6 mg. per cent, Wassermann negative, and total protein 18.6 mg. per cent. Spinal fluid culture was negative and x-rays of the skull were negative.

The clinical diagnosis seemed to lie between an encephalitis or glioma of the brain stem. On October 14, 1948, a pneumoencephalogram was done, replacing 130 ccs. of fluid with air. Films (Figure 1) revealed diffuse enlargement of the entire ventricular system, and an excess accumulation of air

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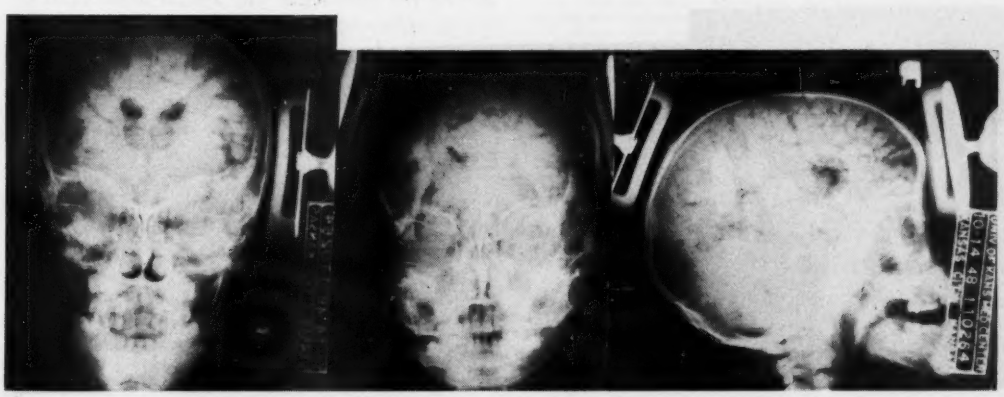

Figure 1. AP, PA and lateral views of pneumoencephalogram done October 14, 1948.

on the surface of the brain outlining shrunken gyri and widened sulci. This was interpreted as being rather severe generalized cerebral atrophy. On this basis, we felt that the child suffered from either a progressive degenerative disease of the brain, or a post-encephalitic atrophy, and that the prognosis was exceedingly poor.

Routine care, gavage feedings, parenteral fluids, and occasional infusions of plasma were given, but no specific therapy was rendered. Within one month the child began to show definite improvement, and in two months was talking and walking with the aid of braces. He was dismissed from the hospital February 19, 1949, and readmitted for evaluation April 16, 1949. At this time he walked quite well without braces, though he had a mild right hemiparesis. Psychometric examination revealed an I.Q. of 71.

Because of our interest in the surprising degree of recovery, pneumoencephalograms were repeated April 30, 1949, six and one half months after the initial air study. Films (Figure 2) revealed an apparent marked improvement in the encephalographic evidence of cerebral atrophy, in that the ventricles were definitely smaller and the subarachnoid markings on the surface of the brain had returned to within normal limits. The child was last seen in the out patient department October 8, 1949, and exhibited only a very minimal right hemiparesis. He walked almost normally, talked well, and had no complaints.

Discussion

The exact diagnosis in this case remains uncertain but the general label of encephalitis, type and cause undetermined, seems justified. The feature to be stressed is the fact that during the early course of the child's illness the clinical picture was that of severe diffuse brain involvement and the pneumoencephalogram revealed evidence of gross atrophy of cerebral substance. Six months later, after a remarkable clinical recovery, a repeat pneumoencephalogram revealed that the cerebral atrophy had, to a large extent, disappeared.

Perusal of the literature reveals such a concept to be almost non-existent. Several authors caution against errors in interpretation of pneumoencephalograms in infancy. Zellweger[4] reports that in the first two years of life, pneumoencephalograms often appear abnormal because of the extreme softness and pliancy of the infant's brain. He examined seven infants whose first pneumoencephalograms closely suggested atrophy of the cortex, but when repeated a year or two later showed normal filling of the subarachnoid space. He recommended repeat air studies after the second year of life. Brines and Lord[5] pointed out errors in predicting subsequent mental age in children under three on the basis of the pneumoencephalogram. They reported six infants with mild cerebral atrophy on the basis of the pneumoencephalograms who subsequently proved to be normal mentally. They did not, however, repeat the pneumoencephalogram to see if such changes persisted, though they did conclude that the pneumoencephalogram in infancy is unreliable. Davidoff and Epstein[3] add a word of caution concerning the interpretation of pneumoencephalograms in infants, warning against the misinterpretation of subdural air for cortical atrophy. Our patient, however, is out of the age group of infancy, and subdural air is not present in the initial air study.

Apparently only one case has been previously reported with pneumoencephalographic evidence of reversible cerebral atrophy, and this was in an infant who, according to Zellweger, makes the pneumoencephalographic findings a bit inconclusive. Wyatt and Carey[6] reported this case, one of congenital neurosyphilis, with pneumoencephalograms at six months showing marked atrophy with dilated sulci and enlarged ventricles. Under antiluetic therapy, marked clinical improvement occurred, and

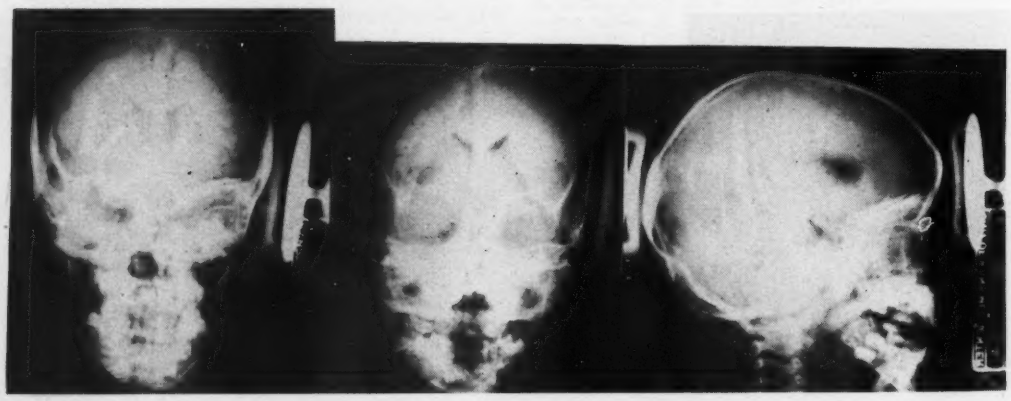

Figure 2. AP, PA and lateral views of pneumoencephalogram in same patient done April 30, 1949.

pneumoencephalograms at 21 months revealed definite improvement in the superficial markings, though the ventricles remained large.

In our patient, the pneumoencephalographic findings after an interval of six months apparently reveal definite evidence of return of gross brain substance as the child recovered from an apparent encephalitis. In the initial pneumoencephalogram, the transverse measurement from the lateral border of the frontal horn to the other in the AP view is 5.3 cm., and in the second air study 4.9 cm. The diameter of the body of the right lateral ventricle, measured from the uppermost limit of the septum pellucidum to the infero-lateral floor, is 1.6 cm. in the first pneumoencephalogram. The same measurement in the second air study is 1.0 cm. The widest sulcus on the later view of the first encephalogram measures .7 cm. and on the last one .1 cm.

It is recognized that one must be cautious in interpreting the surface markings on the encephalogram as being positive evidence of cortical atrophy. The size and shape of even normal spaces vary within wide limits. The frequent experience of different degrees of subarachnoid filling for no apparent reason is recognized by all. It is noted that in our second pneumoencephalogram cortical markings are seen only on one side. Whether or not this is an indication of so-called adhesive arachnoiditis may be argued. Some amount of subdural air may also be seen along the falx in the second air study. However, the positive difference in the size of the ventricles, as well as the marked differences in the surface markings, should be readily recognized.

One must also be aware of the fact that the ventricles may alter in size in a matter of hours after a pneumoencephalogram is performed. Schatzki, Baxter, and Troland[7] pointed out that films repeated on the following day after pneumoencephalography revealed enlargement of the ventricles, as compared to films taken on the first day, in 38 per cent of patients. However, the technical procedure of the two air studies in our patient was identical, with films being taken immediately upon completion of injection of air. The same views, utilizing the same roentgenographic technique, were taken. On both occasions, the total available amount of spinal fluid was withdrawn in 10 cc. quantities, and replaced with an equal volume of air. Unfortunately, the total measured quantity of fluid obtained on the second air study was not recorded.

This report does not intend to lead one to believe that actual regeneration of nerve cells took place. We of course have no microscopic sections showing the type or degree of cell damage that had occurred. Undoubtedly, recovery of partially damaged elements did occur, and on the basis of the pneumoencephalogram, the so-called cerebral atrophy was, at least in some degree, reversible. Actually, it might be that if pneumoencephalography were often performed at the termination of the acute phase of encephalitis, atrophy would be a frequent finding. It is possible that follow-up air studies months later might then reveal that these pneumoencephalographic findings are often reversible. Obviously there is rarely indication for such a procedure, so it is hoped that this report may stimulate future reports to clarify this point.

Summary and Conclusion

The pneumoencephalographic findings of moderately severe diffuse cerebral atrophy usually justify the conclusion that severe permanent brain damage has taken place, and the prognosis for much clinical recovery is poor. These encephalographic findings are usually considered permanent. We report this case in which the pneumoencephalographic finding of cerebral atrophy was reversible. Clinical recovery took place, as well as a marked improvement in the gross structure of the brain as shown on pneumoencephalogram.

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Prolapsing Redundant Gastric Mucosa*

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In the Department of Radiology at the University of Kansas School of Medicine we have only within the last few years become definitely conscious of prolapse of gastric mucosa into the duodenum as a clinical entity. We are convinced it is a condition that can be diagnosed only by the roentgenologist because the clinical symptoms are varied and not classical of any pathological process. Judging from reports by others it is being diagnosed progressively more frequently and occasionally the diagnosis is proved at the operating table. There are still those who believe that a diagnosis of prolapsing mucosa is without clinical significance[11] and may be merely a confusing factor masking other causes of bleeding such as a duodenal ulcer[25] or a palpable mass[26] of antral carcinoma.

Prolapse of gastric mucosa was described by Schmeiden[30] in 1911, 40 years ago. The condition has been described at intervals by Forsell,[8] Eliason and Wright,[6] Meyer and Singer,[17] Pendergrass and Andrews,[21] Bohrer and Copelman,[3] Archer and Cooper,[2] Melamed and Hiller,[15] MacKenzie, Macleod and Bouchard,[14] and others.[19, 22, 16, 31, 4] Scott[28] stimulated new interest by his excellent article in 1946. More recently articles have appeared by Hawley, Meyer and Felson,[10] Nygaard and Lewitan,[20] Ferguson,[7] Appleby,[1] Cove and Curphey,[5] Rudner,[24] Moon and Speed,[18] and Udaondo, D'Alotto and Cabanne.[29]

If there is any question as to the correctness of such a diagnosis as prolapsed gastric mucosa it should be dispelled by the cases reported which have been proved by operation. Rudner,[24] Hawley, Meyer and Felson,[10] Pendergrass and Andrews[21] and many others, including ourselves, have seen cases come to operation in which the diagnosis was confirmed.

Symptoms and Signs

There is no classical group of symptoms that will lead to a diagnosis of prolapsed gastric mucosa. The symptoms commonly are those that suggest an ulcer. The patient may complain of epigastric discomfort, a burning sensation in the stomach, a sense of fullness or pain suggesting pylorospasm or pyloric obstruction and there may be occasionally nausea and vomiting. It is often associated with hyperacidity, but not always. In a few cases there is a history of gastro-intestinal bleeding. Rarely a mass may be palpable. In one of our cases a mass was felt which led to a diagnosis of malignancy of the antrum. The patient refused operation and now, five years later, has the same x-ray findings. The mass is inconstant. Obviously the diagnosis of malignancy was wrong. Cases have been described in which the mass could be reduced by manipulation, much as a hernia may be reduced with the palpating fingers.

Roentgen Findings

The classical descriptive terms used by writers in describing the duodenal bulb are a "mushroom," "umbrella," or "jockey-cap" appearance. With an outburst of modernism, one writer has called it the "atom bomb" appearance (Figure 1). The loose

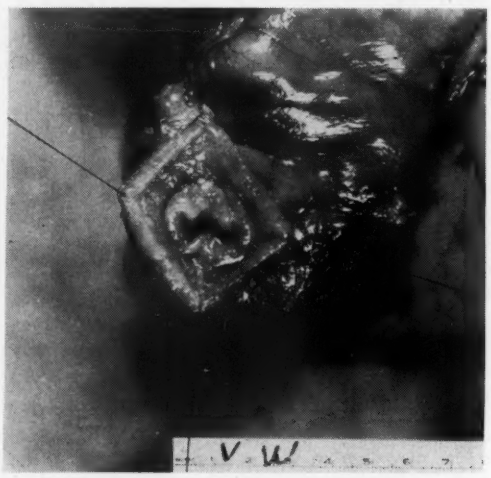

Figure 1. Case 2. Open end of duodenum showing prolapse of gastric mucosa through the pyloric ring.

mucosa of the antrum slips through the pyloric canal to fill the base of the bulb. The base is concave. The free mucosa presents a "crumpled silk" pattern. In the pylorus the mucosal folds stand out as isolated parallel strands running into the bulb as multiple opaque streaks. There may be such slight mucosal herniation that it will be overlooked, or it may be transient and not observed every time the stomach is examined. The other extreme may be a filling defect due to a constant mucosal herniation that amounts to an intussception, in which case there is a permanent defect and a palpable mass that may lead to a diagnosis of antral carcinoma. Such a case has recently been seen and reported to us by Drs. Kubin and Hiebert.[13]

There is no disturbance in peristalsis in the average case. The duodenal bulb retains barium well and is not irritable. In our cases there was no six

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hour gastric retention. Eliason and Wright,[6] in 1925, described a pedunculated tumor or polyp located in the antrum that "is swept or carried into the pylorus by a peristaltic wave and produces a typical ball valve syndrome attack of pain." This, in our experience, is even more rarely seen than is mucosal herniation. It should be differentiated by the circumscribed nature of the mass in the duodenum.

Etiology

Many theories have been suggested to explain the etiology of this condition. It seems logical to conclude that the attachment of the mucosa of the antrum to the underlying tissue is not firm. When this is associated with excessive motility of the muscularis it is not unreasonable to assume that excess mucosa in the antrum might be forced through the pylorus. It also seems reasonable to assume that, after a period of time, fixation of this loose tissue in the pyloric canal may result in edema, congestion, local gastritis and, at the point of constriction, even a palpable mass.

Scott,[28] after reviewing anatomy texts and studying 126 stomachs at autopsy, concluded that prolapse results from an excess and abnormal mobility of the pyloric mucosa on the muscularis. He believed that "the structural conditions necessary for the development of a prolapse are inherent in the walls of the normal stomach, but a prolapse occurs only after the fibers in the flexible submucosa have been stretched and loosened by abnormal gastric peristalsis, which in turn is irritated by neurogenic or chemical stimuli, or a combination of both."

The opinion that there is independent movement of the mucosa was offered by Forsell[8] in 1923. By pictures of anatomical preparations, series of roentgenograms and photographs of the digestive tract of living subjects, he rather convincingly demonstrated that "the muscularis mucosae forms the special contractible organ of the mucous membrane which, being attached to this one and to the submucous layer, is able to displace the mucous membrane in all directions by means of transversal, longitudinal and oblique fibers."

Golden[9] also emphasized the free movement of the mucosa of the stomach over the muscularis. He described a change in mucosal direction from transverse to parallel with the long axis of the stomach when antral systole occurs. There is a stretching mechanism he considers normal. The failure of the stretching mechanism may account for herniation of prepyloric mucosal folds through the pylorus. Golden also expressed the opinion that pyloric canal thickening, a condition that must be considered with the subject of mucosal herniation, is related to antral gastritis. Schindler[27] has verified by gastroscopic observations the formation of radial folds with antral peristalsis. Eliason and Wright[6] theorized that as a result of chronic, low grade inflammation the mucosa becomes hypertrophied. This hypertrophied mucosa is then pushed toward the pyloric canal by peristalsis and may be pushed through the pylorus in some cases.

Rees[22] believed that narrowing of the pyloric diameter precedes the actual change in the gastric mucosa and that the narrowing is followed by hyperperistalsis of the stomach in its effort to force its contents through the smaller opening. This action then loosens the attachment of the mucous membrane to the muscularis in the constricted area. Archer and Cooper[2] report a case in which at operation "on passage of the examining finger through the pylorus a very tight, firm ring was found and the mucous membrane in the prepyloric portion of the stomach was less firmly attached than usual. When the duodenal bulb was incised, there was a protrusion of gastric mucosa through the pylorus resulting in somewhat the appearance of external hemorrhoids about the anus."

This case tends to verify the observation made by Rees. Bralow and Spellberg[4] cite cases of prolapsed mucosa due, in their opinion, to gastritis. They say, "A benign peptic ulceration located at the base of the duodenal bulb or in the prepyloric area may, in the process of healing, produce a local gastritis. This inflammation may lead to a localized hypertrophy of the gastric rugae of the prepyloric areas which eventually may prolapse through the pylorus into the base of the bulb at a time when no other sign of the marginal ulcer may be present."

Melamed and Melamed[16] reported recently four cases of right heart failure with gastrointestinal symptoms and evidence of prolapse of gastric mucosa into the duodenum. It was their suggestion, after study of these cases (two found at autopsy), that markedly redundant edematous and mobile gastric rugae associated with edema of the mucosa may provide an adequate basis for increased mobility of the gastric mucosa and subsequent prolapse into the duodenum. They suggest that in many cases of congestive heart failure gastric symptoms may be due to this condition.

Incidence

Wellens and Spyckerelle,[31] in 1949, were able to find only 60 cases reported in the literature. These authors estimate mucosal prolapse is seen in five per cent of stomach examinations. Scott[28] analyzed 19,228 hospital admissions. Of this number 1,346 upper gastrointestinal examinations were made. The pathology revealed was as follows: 0.96 per cent gastric ulcer, 24.1 per cent duodenal ulcer, and 1.04 per cent redundant gastric mucosa. Redundant mu-

cosa was more commonly seen than was gastric ulcer. Cove and Curphey[5] found 3.38 per cent in 650 consecutive examinations.

In our Department of Radiology we have diagnosed 20 cases of mucosal herniation during the year 1950. In this period of time 2,312 stomachs were examined of which 16 per cent were diagnosed duodenal ulcer, two per cent gastric ulcer and 0.86 per cent prolapsed gastric mucosa. Three patients have been operated upon and the diagnosis proved. When we consider the relatively small number of cases reported in the literature as compared with one to five per cent seen by some investigators in routine examinations of the stomach, we wonder if the condition is being accurately diagnosed by those who see so many, or if we who see so few are missing some of them.

Differential Diagnosis

If there is a palpable mass or constant defect in the pylorus, as in the case described by Kubin and Hiebert,[13] a diagnosis of carcinoma of the antrum of the stomach will likely be entertained. In our case the defect was not so marked, but the antrum could be rolled beneath the palpating fingers, leading us to an erroneous diagnosis of carcinoma. One has to wonder what we called this condition 10 years ago when we were not so keenly aware of its possibility. I think undoubtedly chronic duodenal ulcer was diagnosed because of bulb deformity. Now, unless a crater is visualized, a diagnosis of duodenal ulcer is not justified if the classical picture of herniation of mucosa is seen. There have been reports of an ulcer and mucosal prolapse in the same case.[15] Also malignant disease has been found associated with mucosal prolapse.[23]

Hypertrophy of the pyloric muscle was described by Kirklin and Harris[12] in 1933. They reported 81 cases seen in a five-year period. It was their conclusion that this condition was usually associated with duodenal, gastric or cholecystic disease. They noted that the stiff pylorus tended to invaginate into the duodenum. They did not describe with this condition the central umbrella defect so characteristic of mucosal invagination. A definite mass was not felt in their cases.

Treatment

The treatment of prolapse of the gastric mucosa is usually symptomatic and conservative. In the average case the symptoms are relatively mild and can be controlled by medical therapy. If bleeding is not readily controlled, if symptomatic treatment for ulcer without definite demonstration of an ulcer has been unsuccessful, if there is partial obstruction at the pylorus or if a mass is palpated and a defect is seen which remotely resembles a carcinoma, operation is the treatment of choice.

The operative treatment of prolapsed gastric mucosa has not been standardized. Several operations have been described. These are partial gastrectomy, pyloroplasty, pyloroplasty (Horsley, Finney or Heineke-Mikulicz) with excision of redundant mucosa and gastrotomy with excision of redundant mucosa. It is probable that a single technique is not suitable for all cases.

Three cases that have been treated by operation at the University of Kansas Medical Center are here reported briefly.

Case 1. T. O., a female aged 36, entered the University of Kansas Medical Center January 1, 1950. Eight months after the birth of a child in 1947 she had a severe hemorrhage from the alimentary tract. A diagnosis of duodenal ulcer and possible prolapse of gastric mucosa was made by her local physician. After entering the hospital a splenomegaly was found and the diagnosis of prolapsed gastric mucosa was confirmed. An ulcer was not seen by x-ray examination. There was a slight hyperacidity. A splenectomy was done at the same time the prolapsing gastric mucosa was excised followed by a Horsley type of pyloroplasty. No evidence of ulceration was found at the operation or by the pathologist. There was edema and recent hemorrhage in the gastric mucosa. A diagnosis of Banti's disease was made. To date there has been no recurrence of bleeding.

Case 2. V. W., a male aged 46, entered the hospital on March 6, 1950, with a diagnosis of duodenal ulcer and a history of six episodes of gastrointestinal bleeding since November, 1949. The x-ray examination showed a typical "mushroom" deformity of prolapsing gastric mucosa. There was a definite hyperacidity. At operation a thickening was found at the pylorus with a widening of the first portion of the duodenum. A partial gastrectomy of the Hofmeister type was done. The gross speci-

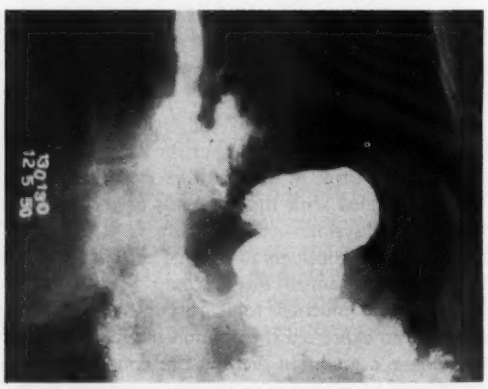

Figure 2. Case 3. Hiatal hernia and hernia of gastric mucosa into duodenum. Mucosal prolapse proved by operation. Source of bleeding not found.

men showed a prolapse of the gastric mucosa (Figure 2). The pathologist reported "section of stomach reveals the mucosa to be densely infiltrated by mononuclear cells. The lymphoid follicles are above and below the muscularis mucosa, some being hyperplastic. The cells in the mucosa are prominent and congested. The subserosa shows numerous foci of recent hemorrhage. Section at the pylorus reveals the mucosa to contain a lesser number of glands, some of which are distorted in the lamina propria. In areas the fibrous tissue extends into the muscle layer as a thickening of the normal architecture. There is no evidence of malignancy or ulceration." This patient has had no bleeding since the operation and is now free from gastric symptoms.

Case 3. F. O., a 74-year-old female, entered the hospital December 3, 1950, with a history of hematemesis and melena for three days. She received two transfusions before entering the hospital. X-ray showed a large diaphragmatic hernia and a prolapse of the gastric mucosa. The hernia was repaired and a pyloroplasty of the Horsley type was done. The source of the bleeding was not found. She made a good recovery and there has been no bleeding since the operation. At the time this is written, only six weeks after the operation, the final result cannot be determined.

The three patients recorded above were treated primarily for bleeding from the upper gastrointestinal tract. In none of the three are we sure that the bleeding was from the prolapsed gastric mucosa. The pathologist's report in Case 2 indicates that the prolapsed mucosa was probably the source of the bleeding.

More than 40 recorded cases of prolapsed gastric musoca have been treated by operation and this type of therapy now appears to be well established in selected cases.

Conclusions

1. Prolapsing gastric mucosa is not as rare as the few cases reported seem to indicate.

2. The diagnosis must be made by accurate roentgen observation. Symptoms are not sufficiently well standardized to justify a clinical diagnosis.

3. The average case presents the "mushroom" deformity of the bulb considered characteristic of mucosal herniation. Occasionally the deformity may not be distinguished from carcinoma.

4. The cause of prolapse of the gastric mucosa has not been determined. The anatomical demonstration that the mucosa may move independently of the underlying structure of the stomach when associated with hyperperistalsis seems to us sufficient evidence that the condition may occur without presupposing some associated serious pathological process. Gastritis of the antral mucosa may be found at operation. We raise the question as to whether the gastritis is cause or effect.

5. Surgical treatment is indicated when there is gross or continued bleeding, when carcinoma is suspected, when there is partial pyloric obstruction with vomiting, and when medical treatment has failed to relieve symptoms.

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A Re-evaluation of the Surgical Treatment for Duodenal Ulcer as Based on Experimental and Clinical Observations

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Inasmuch as surgeons are still called upon to operate on patients with duodenal ulcer and its complications, it is mandatory that a surgeon perform an operation which meets the criteria for satisfactory results. These criteria must be based on sound experimental investigation of the physiology of the upper intestinal tract, as well as the experience gained in the study of patients operated upon for ulcer.

For well over a half century, surgeons have been operating upon the stomach and duodenum for the treatment of peptic ulcer. Until recently, most of the operations were employed purely on an empirical basis with very little knowledge of the physiological concepts determining a good operation for ulcer. It has long been known that the most important factor in the genesis and production of peptic ulcer is the gastric acidity with the associated pepsin, and it follows that most surgical procedures today are designed to abolish, or greatly diminish, acid secretion.

The operations employed, however, vary primarily in their attack of the various phases of gastric secretion: the cephalic phase, the gastric phase, and the intestinal phase. Granting that the gastric acidity is the most important factor in ulcer genesis, there have also been found many precipitating or abetting influences to the ulcer diathesis. Obviously if acid were the only factor in operation, all stomachs containing acid would probably present ulceration. This, of course, is not the case, and the hypothesis of mucosal predisposition must be considered. Many of these predisposing influences have been studied and have, as a common denominator, the element of mucosal ischemia and include such conditions as arterial thrombosis or embolus, arterial spasm, hemoconcentration, venous statis due to portal obstruction, fatty embolism, and many such influences. Because of the many and diverse predisposing conditions, it is natural that the operative treatment has concerned itself primarily with the ablation of the acid factor in the ulcer genesis and it is the background of the primary acid factor which is discussed here.

Early Experimental Investigations

In the early experimental investigations of the ulcer problem, it was necessary to select an animal suitable for ulcer study. Of all the experimental animals, it was found that the dog presented the unique situation satisfying the requirements for this type of investigation. The dog is an animal in which ulceration does not occur spontaneously. This is in contra-distinction to man, in which spontaneous ulceration is common. It was felt that if ulcers could be produced in dogs with regularity, then operations could be compared as to the protecting ability against such uniform ulcer production.

Mann and Williamson[1] had produced peptic ulceration in dogs by the transplantation of the alkaline secretions to lower levels of the small intestine, thus spatially separating the acid from the alkaline secretions. Their experimental preparation, however, did not produce a uniform and completely predictable occurrence of ulceration. Because of the capacity of histamine to specifically stimulate the parietal cells of the stomach to secrete increased amounts of hydrochloric acid, it naturally followed that histamine would be employed in an attempt to produce ulceration in animals. Whereas several investigators had been unsuccessful in this, it was not until Code and Varco[2] incorporated histamine in a beeswax mixture that experimental production of ulcers in dogs was uniformly accomplished. The histamine-in-beeswax mixture injected intramuscularly once daily into dogs will produce a high and sustained elevation of gastric acidity for a period of 24 hours. When it is injected daily over a period of 40 days, the occurrence of ulceration, predominantly duodenal, is a uniform and regular occurrence.[3] This useful tool became universally popular for the study of ulcer genesis and treatment.

Type of Resection

Following this, the earliest significant piece of research concerning the ulcer problem was the natural employment in dogs of the many operations ordinarily performed on patients, in order to compare their protective ability against the histamine-provoked ulcer. The contribution of Lannin[4] is significant in which seven series of experiments were carried out (see Figure 1). In the first series a group of dogs were subjected to the operation of gastro-jejunostomy. After a month's convalescence, these dogs were begun on a daily series of histamine-in-beeswax injections. All four dogs so treated developed large perforating jejunal ulcers. Perforations

were noted in two dogs after three and 13 daily injections, and in two dogs the large ulcers were noted after 40 histamine injections.

This series of experiments clearly indicates that gastro-jejunostomy affords no protection whatsoever against the histamine-provoked ulcer. As a matter of fact, from human experiences and many subsequent experiments in dogs, gastro-jejunostomy hastens the occurrence of ulceration. It has been shown that patients undergoing gastro-jejunostomy for whatever cause have a 20 to 40 per cent chance of developing stomal ulceration, clearly demonstrating that gastro-jejunostomy no longer enjoys the enthusiastic following of two to four decades ago.

The next series of experiments concerned the popular operation of antral excision or small gastric resection. Four dogs were subjected to such an operation and after 35 to 40 daily injections of histamine-in-beeswax all dogs presented large jejunal or stomal ulceration, again indicating that small gastric resection does not protect against the histamine-stimulated ulcer in dogs. From further experimentation this fact is undoubtedly due to the failure of excision of enough acid-bearing tissue of the stomach. It is also apparent that excision of the antrum with its presumable hormone, gastrin, according to the Edkins' phenomenon is not sufficient for the treatment of ulcer.

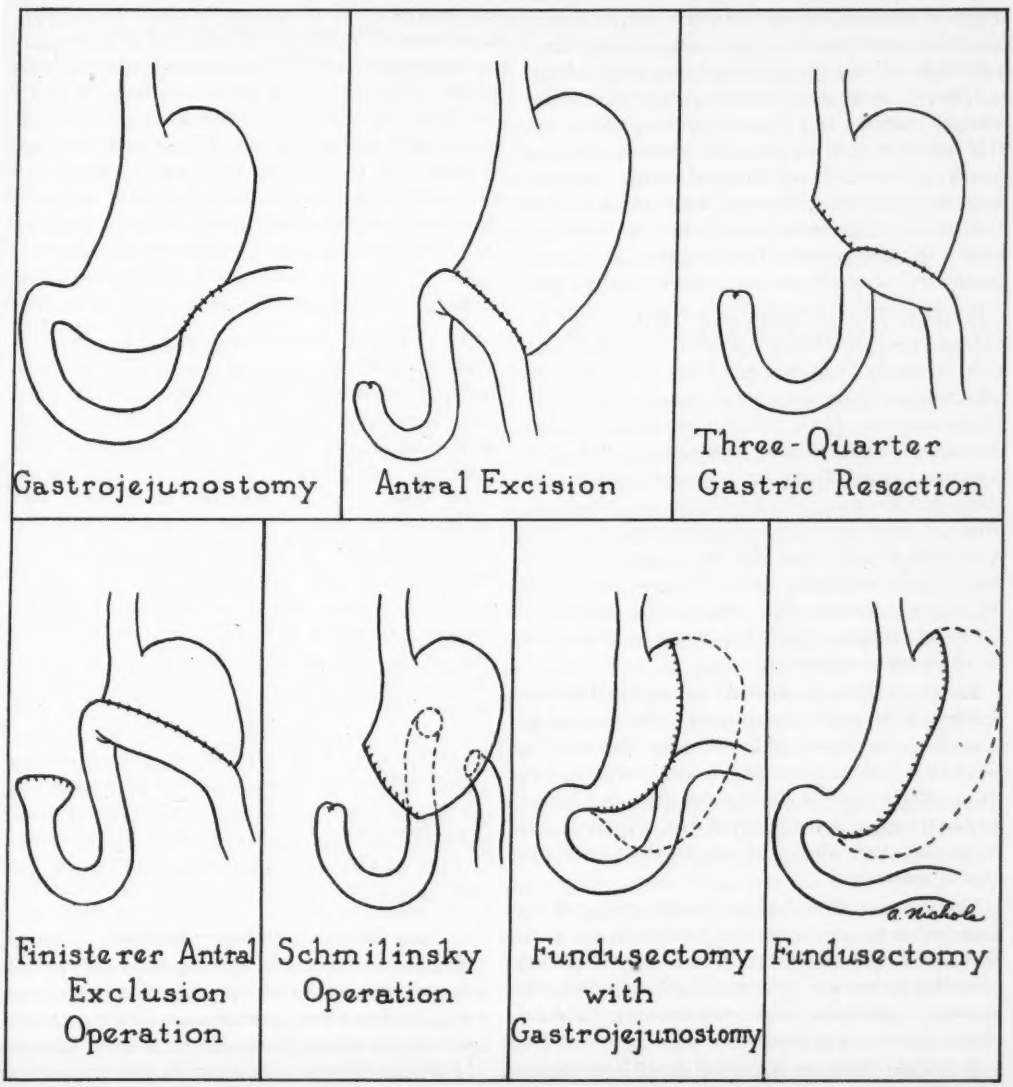

Figure 1

The third series of experiments utilized the three-quarter gastric resection. Eight dogs were subjected to such an operation, and all received 40 daily injections of histamine-in-beeswax, after which time no ulceration whatsoever was noted in any of these dogs. This showed clearly that it is necessary to excise three-fourths of the stomach, or that much of the acid-secreting tissue, to protect against the maximum stimulation of histamine-in-beeswax. The refinements and attributes of the three-quarter resection will be discussed more in detail subsequently.

The previously popular antral exclusion operation described by Finsterer was employed in the fourth series of experiments. This operation connotes an extensive resection, except for the antrum which is merely excluded from the gastro-jejunostomy. Four such dogs received histamine-in-beeswax injections, and after 32 to 40 daily injections there were noted erosions, gastritis, and jejunitis, without ulceration. This indicates that the extended resection is satisfactory, but not as beneficial as the series three experiment. Also this tends to put discredit on Edkins' phenomenon and implies that the antrum is not important in ulcer genesis. This, however, is not necessarily true as will be shown in later investigations.

In series five of these early investigative researches, the Schmilinsky operation was employed. This operation combines not only antral excision but complete intragastric regurgitation of the alkaline secretions from the proximal loop into the stomach. The Schmilinsky operation enjoyed great popularity, especially on the continent, but has since fallen into discredit. The four dogs in the early experiment receiving histamine-in-beeswax all developed large jejunal ulcers after two, eight, 11 and 40 days, clearly indicating that this operation affords no greater protection than simple antral excision or gastro-jejunostomy. Indeed, it tends to hasten the development of ulceration.

The sixth series of experiments employed fundusectomy with gastro-jejunostomy. The four dogs treated in this manner followed with histamine-in-beeswax injections developed duodenal erosion, gastritis, etc. In this operation a moderate number of acid-secreting cells are removed, but apparently this is not sufficient when it is compared to the three-quarter resection.

The seventh series of dogs is similar to that of the sixth in that fundusectomy was performed, but without gastro-jejunostomy. Four such dogs receiving histamine-in-beeswax were sacrificed at 40 days, one showing a duodenal ulcer, two showing duodenal erosion and one a gastritis.

It is clear from the above series of experiments early in the investigative work on the duodenal ulcer problem that only one operation, or modifications of it, regularly protected dogs from the maximum stimulus of histamine injections, that being the three-quarter gastric resection.

Role of the Length of the Afferent Duodenojejunal Loop

Because it was necessary, following these early experiments, to determine what refinements or what factors were operating in the three-quarter resection to produce such dramatic protection, further series of experiments were outlined. The following are the significant works. Since it had already been significantly demonstrated that three-quarter resection was necessary, it remained to be shown whether a long or short proximal, or afferent, duodenojejunal loop played a part in such protection.[5] Accordingly three series of dogs were all subjected to three-quarter resections. In Series I, however, there was employed a short proximal loop measuring 12 to 15 cm. from the inverted duodenum. Histamine administration was also employed in Series I. In Series II seven dogs were subjected to three-quarter resections and histamine-in-beeswax injections, in which there were employed long proximal loops measuring 27 to 78 cm. in length. In Series III four dogs received 75 per cent resections with long proximal loops without histamine-in-beeswax administration.

All 11 dogs in Series I demonstrated no ulceration after 40 daily injections of histamine-in-beeswax, indicating a clear protection to the maximum histamine stimulation. In Series II all of the seven dogs with three-quarter resections and long loops with histamine administration developed stomal ulcerations, three of which were free perforations with subsequent peritonitis; a 100 per cent incidence of stomal ulceration. In Series III where the long loop was employed with extensive gastric resection in which no histamine was given, there were two spontaneous occurrences of stomal ulceration, indicating that in the dog, in which spontaneous ulceration does not occur normally, spontaneous ulceration can be made to occur if a three-quarter resection with a long loop measuring 78 to 144 cm. is employed. Thus this early work definitely demonstrated that a short loop is far superior to the employment of the long loop, as a refinement to the standard three-quarter gastric resection. It has also been found that it is not necessary to excise a duodenal ulcer in the performance of a gastric resection.

Criteria of Satisfactory Operation

Because of the above investigations, as well as many other studies, it was possible then to establish certain criteria for the performance of a satisfactory operation for ulcer.[6] These may be listed as follows: (1) Seventy-five per cent resection with excision of the entire lesser curvature so that a sufficient amount

acid-secreting mucosa is removed from the patient; (2) complete excision of the entire antral and pyloric mucosa; (3) employment of a short afferent or proximal loop. A short loop must be defined as one in which the anastomosis is made at or just proximal to the ligament of Treitz. Any anastomosis performed distal to that point must be considered a long loop. This implies that, to carry out a short loop anastomosis, the duodenum and jejunum must of necessity be brought up through the mesocolon in a retrocolic fashion for anastomosis. Conversely, any loop that is brought up in an antecolic fashion with the anastomosis anterior to the transverse colon must necessarily be called a long loop.

Attributes of the Short Proximal Loop

Much investigation has been carried out in which the various attributes of the short loop have been more clearly defined.[7] It was necessary to determine why a short loop was superior to the long loop. The most apparent virtue of a short loop is the matter of distance needed for the alkaline secretions to reach the anastomosis for neutralization of the acid. The shorter the distance the less time involved for neutralization to take place. In other words, the fire station is closer to the fire in the short loop anastomosis. Similarly, the transit time is much reduced with the short loop.

The second significant experimental contribution to efficacy of the short loop incorporated studies of the differential titratable alkalinity at various loops of the small intestine. There is a constant interchange of ions between the lumen of the bowel and the blood stream at all levels of the intestinal tract. The alkaline secretions from the pancreas and liver constantly undergo such changes as they progress down the intestine, and it has been shown that the titratable alkalinity decreases in proportion to the length of the bowel traversed. Therefore, the shorter loop would deliver increments of higher titratable alkalinity for neutralization of the acid at the anastomotic site.

The third important factor relating to the efficacy of the short loop is the secretin factor. It has been well shown in animals that if acid of gastric juice or tenth-normal hydrochloric acid is delivered to the mucosa of the small intestine, secretin is released.[8] This hormone from the mucosa is released to the blood stream which, in turn, stimulates the pancreas to secrete the alkaline pancreatic juice. It has been shown, in addition, that the various levels of the intestine vary in their sensitivity to acid in the release of secretin. In other words, the higher the small intestine, as far up as the duodenum and pylorus, the greater the sensitivity to acid, the greater the secretin released and the greater the pancreatic secretion. Consequently, if a short loop anastomosis is employed where the acid from the stomach is deposited high in the small intestine, the greater will be the secretion of pancreatic juice for subsequent neutralization of the acid at the anastomosis.

The fourth attribute of the short loop anastomosis is the ill-defined factor of tissue susceptibility to ulceration. The lower reaches of the small intestine are apparently more susceptible to ulceration than are the more cephalad segments. Accordingly, a short loop gastric anastomosis with the small bowel would present less susceptibility to ulceration. This last factor may be a result of the first three named, yet it may indicate a specific immunity or susceptibility as the case may be. These four factors clearly demonstrate that a short loop is superior to a long loop anastomosis. It naturally follows that a Billroth I type of gastric resection, in which the divided duodenum at the pylorus is anastomosed end-to-end with the gastric pouch, would completely satisfy these requirements in that no loop at all would be proximal to the anastomosis. Technically, however, it is impossible to do a Billroth I operation on all patients with ulcer because of spatial difficulties in securing the gastric pouch in connection with the divided duodenum without tension. This can be done in the long, lanky type of individual but not in the broad-chested type of patient.

Clinical Investigations

The above criteria are adequately documented in the experimental literature and, indeed, are substantiated by experiences in clinical surgery. Perusal of the surgical literature concerning the results of operations in patients again establishes the above-named experimental observations. For instance, of 416 cases consecutively operated upon for peptic ulcer at the University of Minnesota in which the above criteria were rigidly adhered to, there occurred only three instances of recurrent stomal ulceration, an incidence of less than one per cent.[9] One additional patient has had recurrent bleeding without x-ray demonstration of ulceration. Inclusion of this case would still make for a recurrence rate of less than one per cent.

Because of the experimental background, it was felt that even these few recurrences should be explained. Accordingly, two of the three patients were re-explored. It was found in the first, whereas it was thought at the first operation that a three-quarter resection was carried out, this was not the case. Actually, less than a three-quarter resection had been performed, presumably because the greater portion of the stomach was tethered high underneath the diaphragm as one so often sees in the short, broad-chested individual in whom the stomach assumes a flat or transverse contour. At re-

operation in this patient further resection of the stomach was done. In the second case in which reoperation was carried out, it was found that the proximal loop was not as short as experience has shown to be necessary; this was corrected, as well as excision of slightly more gastric tissue. These recurrences, then, do not incriminate or discredit the criteria set up but only substantiate them. It is reasonable to assume that if the criteria were met faithfully in every case, the incidence of recurrence should be zero.

Further correlaries in human experience to experimental evidence are shown by the study of other reports in surgical literature. The incidence of recurrent ulceration varies in large series from seven to 15 per cent. Kiefer,[10] reporting the results of gastric resection at the Lahey Clinic, noted a recurrence rate of 11.4 per cent following gastric resection for duodenal ulcer, in cases where the long proximal loop was employed. Rienhoff[11] reported an incidence of recurrence of 11.3 per cent. It is likely that the addition of 20 to 40 cm. of jejunum to the proximal loop, when antecolic anastomoses are performed, probably accounts for the increased recurrence rate of stomal ulceration. Again a short loop must be defined as one in which the anastomosis is retrocolic and at or just proximal to the ligament of Treitz.

Operations on the Autonomic Nervous System

It is pertinent to discuss the operations performed on the autonomic nervous system for the treatment of peptic ulcers. Similar to the other procedures, these operations are designed primarily for the ablation of the acid factor, the particular distinction being that the cephalic phase of gastric secretion is considered to be the most important. Experimental investigation has been carried out for many years prior to their recent employment in patients. In his early physiologic studies on gastric secretion, Pavlov[12] showed that in the vagus denervated gastric pouch of a dog the cephalic phase is eliminated by the denervation, although the pouch still had the capacity to secrete acid in the gastric and intestinal phases of gastric secretion.

More recently vagus interruption has been shown physiologically to be important in the diminution of the night hypersecretion of acid so often seen in patients with duodenal ulcer.[13] Concerning the relation of the sympathetic nervous system to the peptic ulcer genesis, studies of significance show that coeliac ganglionectomy in dogs predisposes markedly to the histamine-provoked ulcer.[14] Dogs subjected to postganglionic resection will develop perforating ulcers after only two or three injections of histamine-in-beeswax. When vagotomy is employed in animals, there is demonstrated very little if any protection against the histamine-stimulated ulcer,[15] as one might expect, but it is interesting to note that when the two operations are employed simultaneously, that is, postganglionic resection of the sympathetic supply to the stomach together with transthoracic vagotomy, a remarkable protection occurs against the histamine-provoked ulcer in dogs.[16] It is reasonable to assume that the side effects, such as gastric dilatation and stasis, attending complete denervation would create serious problems in patients.

Vagotomy, without complemental procedures, performed in many patients over the country has enjoyed at least a transient enthusiastic response, but it has been quite well shown, in the few years of its employment, that the increasing number of recurrences and the undesirable side effects preclude it from being the best operation for peptic ulceration. The use of small gastric resections with vagotomy or gastro-jejunostomy with vagotomy have not been employed for sufficient periods of time on enough patients to warrant prediction as to their true efficacy as a surgical treatment for ulcer.

In conclusion a sampling of the significant experimental investigations, as well as experience gained from patients operated upon for peptic ulcer, has been presented which establishes clearly certain criteria for satisfactory operation. It is reasonable to expect that if these criteria are faithfully met in every operation designed to diminish the acid factor in peptic ulceration, the recurrence of such ulceration would be an extremely unlikely possibility. These criteria include, primarily, a three-quarter resection of the stomach, excision of the entire lesser curvature and antrum with pyloric mucosa and, three, the employment of a short, afferent duodenojejunal loop with retrocolic anastomosis.

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Inhibition by Antihistaminic Compounds of Growth of Pathogenic Fungi: The Effect of Chlor-Trimeton Maleate in Vitro

Jeannette S. Carter, Ph.D., and Richard L. Sutton, Jr., M.D.*

Kansas City, Missouri

Pyribenzamine ointment was observed clinically to be beneficial in cases of tinea pedis, Carson and Campbell[1] reported, so these investigators tested the compound and found it to be effectively inhibitory in vitro to the growth of various fungi pathogenic for man. Following their studies, we chose Chlor-trimeton maleate** [1-(p-chlorophenyl)-1-(2-pyridyl)-3-N, N-dimethylpropylamine maleate] for similar tests. Clinically this drug given by mouth has appeared to us to be especially satisfactory in patients whose allergic symptoms were of a sort wherein antihistaminic therapy was indicated.

Tubes of Mycophil Broth[R] (Baltimore Biological Laboratory) containing Chlor-trimeton maleate in concentrations of 0.1, 0.25, 0.5, 0.75, and 1.0 mg/ml at pH 6 and at pH 8 were inoculated with two drops of a saline suspension of Candida albicans, Microsporum lanosum, Trichophyton rubrum, and Trichophyton mentographytes. Controls were run with inoculations into plain Mycophil Broth[R] at pH 6. All strains were recently isolated from patients seen in the practice of dermatology. Cultures were examined at 24-hour intervals for 12 days.

Results indicate that Chlor-trimeton maleate at least inhibits the growth in vitro of the four fungi tested. Temporary inhibitory action for 24 hours was obtained with all four fungi in a concentration of 0.25 mg/ml at pH 8. Growth of Microsporum lanosum and Trichophyton rubrum was prevented in a concentration of 0.5 mg/ml at pH 8. No growth of the four fungi tested occurred in concentrations of 0.75 mg/ml at pH 8. The organisms grew better at pH 6 than at pH 8 in plain Mycophil Broth.[R] Chlor-trimeton maleate did not prevent growth of any of these fungi in Mycophil Broth at pH 6, even in a concentration of 1.0 mg/ml.

We are now testing in vivo alkaline ointments containing 0.25 per cent Chlor-trimeton maleate. Clinical results are as yet still being collected.

*From the Department of Dermatology and Syphilology, University of Kansas Medical Center.

**Supplied by the Schering Corporation, Bloomfield, New Jersey.

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The Treatment of Congestive Failure

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The treatment of congestive heart failure has changed during the past five years. The majority of the changes have been for the better, allowing liberal fluid intake, reducing sodium intake, and freer use of mercurial diuretics, but the general adoption of such ideas has tended to result in the establishment of treatment patterns or systems. The average patient can be compensated by such measures; however, there are others who cannot. The approach of digitalization by the large, single oral dose, of the daily administration of mercurials, of the strict curbing of sodium are measures which are effective in many, but more general awareness of the limitations is needed.

1. RAPID DIGITALIZATION.

It is the rare patient who needs his total digitalis effect within six hours. The routine use of 1.2 mgm. of digitoxin as a single initial dose is more dramatic than therapeutic. The choice of digitalis preparation is unimportant; remember that the multitude of products available vary in *speed* of action, not in *method* of action. Among many physicians there has been a recession from digitoxin to the dried leaf. This has been occasioned by the too enthusiastic use of digitoxin, not because digitoxin has any inherent toxicity not also present in the dried leaf.

The term "digitalize" means to give enough of the drug "to obtain the desired therapeutic effect or until toxic symptoms occur." The desired therapeutic effect is compensation of the failing heart, and any arbitrary rule as to what is the necessary amount should not keep the physician from giving less or more. Consider the frequently quoted system of 1.2 mgm. of digitoxin as but a warning that you have given a fair quantity of the drug, but unless you have achieved the desired effect (beginning compensation), continue giving the drug until the effect is reached or until toxic symptoms occur. For example, in the average case a base dose of .6 mgm. of digitoxin by mouth may be given, then every six hours give .2 mgm. additional dose. Continue this additional .2 mgm. until you reach the desired effect or, if toxic symptoms occur, stop the medication until the symptoms pass, and then place on a daily dose. This technique permits you to explore cautiously the given patient's tolerance, and if you exceed this tolerance you will do so by only .2 mgm. of digitoxin. The range of requirement for therapeutic effect can vary from .8 to 3.0 mgm. of digitoxin, for example.

This logically leads to the question, "What is the daily maintenance dose?" The answer is, "Enough to maintain digitalization." Such an answer is not meant to beg the question but is given to indicate that again there is no rule to govern the daily requirement. For the majority it is probably .15 mgm. daily, but there are many others who will require .1, or .2, or .3 mgm. daily. The technique of *re-digitalization* is little used but should have wider application. For example, if a patient has been digitalized and then placed on a daily ration of .15 mgm., it is a good therapeutic measure, after two or three weeks, to increase the daily amount in an effort to explore the margin of toxicity. If doubling the daily ration to .3 mgm. for four or five days does not result in toxic symptoms, it is apparent that the original .15 mgm. allotment was inadequate. Such a re-evaluation may be done periodically.

2. MARKED SODIUM RESTRICTION

The restriction of sodium is important, but remember that the maintenance of nutrition is vital. A rigid enthusiasm for restriction will not infrequently result in a disagreeable patient, who complains bitterly of the tasteless diet, refuses his tray, and is unable to maintain his nutrition. A moderate restriction of four to five grams of NaCl a day will allow a fairly palatable diet. It is appreciated that occasional cases will need further restriction, and when necessary the intake can be reduced to 200 mgm. of sodium daily; refractory failure warrants intensive restriction.

However, it should be remembered that the mercurial diuretics offer an excellent method of ridding the body of its sodium. The urine of an individual, following mercurial diuresis, will not only be increased in volume but also in the percentage composition of sodium. The mercurials increase the renal loss of sodium per unit of renal water. This fact should be more widely utilized in permitting a greater latitude in regard to the dietary sodium. In an elderly individual, for whom eating represents one of the scant pleasures remaining, it seems wise to allow a palatable amount of salt with the food, and to counteract its edema-formation tendency by more frequent mercurials.

The above statement must be qualified by reference to the dangers of over-enthusiastic diuresis. With the enthusiastic adoption of marked sodium restriction and intensive mercurial diuresis, physicians have been seeing a phenomenon which was formerly limited to such states as Addison's disease

and post-operative patients. This state is characterized by weakness, loss of appetite, leg cramping, loss of interest, and somnolence. These are the symptoms of salt-water depletion. If the diuresis is carried too far, kidney failure and irreversible uremia may occur. When such an event is initiated, the physician must make an about face and give sodium chloride by mouth or vein. Such problems can be avoided if the dosage of the mercurial is kept small and if the patient is questioned following each diuresis for the symptoms above described. The intramuscular administration of .5 cc. of mercuhydrin, for example, will cause a moderate weight loss of two to three pounds, which seems far more physiological than an eight to 10 pound flood, followed by weakness and leg cramps. The patient can be taught to follow his weight at home and to give his own intramuscular or subcutaneous mercurial, when he notes a three or four pound weight increase.

3. BED REST.

Rest for heart disease is probably the oldest and most fundamental treatment. However, rest for a patient with orthopnea and several pounds of fluid in his legs is achieved with the patient straight up in a chair, and not recumbent. The abuse of bed rest has been decried by many others, and the observation by the patient that the swelling of his legs disappears when he is in bed is still further warning that edema fluid follows the pull of gravity and the flanks and lung bases are the most recumbent areas when we sleep.

Restriction to bed, with the resultant inactivity of the lower limbs, plus the passive congestion in the veins, can quickly and silently result in thrombi formation in the calf veins. The often fatal sequel, pulmonary embolism, probably accounts for the fatal outcome of more patients with congestive failure than does the original offender, the heart. Not infrequently, residual ankle edema will persist until the patient is allowed to walk and then his leg muscles will mobilize the stubborn fluid.

Summary

The treatment of congestive failure has reached the unfortunate state in which the method has become patterned. The doctrine of rigid sodium restriction, intensive mercurial diuresis, and fixed digitoxin program has resulted in excellent therapeutic results for many. However, for many others it has resulted in unpalatable diets, marked salt-water depletion, and unsatisfactory digitalization.

Kansas Medical Society Annual Meetings

1951	1952
May 14-17	May 5-8
TOPEKA, KANSAS	KANSAS CITY, KANSAS

PRESIDENT'S PAGE

Dear Doctor:

The third annual Kansas Medical Society day was held at the Kansas University Medical School on March 5. The society takes over for the day and presents a diversified program to the juniors and seniors on subjects which we think will be of special interest to them.

The program this year consisted of:

Address of Welcome—Franklin D. Murphy, M.D., Dean of the Kansas University Medical School.

Introduction—F. R. Croson, M.D., President, K.M.S.

Prepayment Insurance in Medicine—W. F. Bernstorf, M.D., First Vice President, K.M.S.

Continuing Education of a Physician—H. H. Jones, M.D., Chairman, Committee on Graduate Education, K.M.S.

General Practice Today—George Burket, M.D., Chairman, Committee on Education, Kansas Academy of General Practice, and Mr. Mac Cahal, Executive Secretary, American Academy of General Practice.

The Physician's Public Relations—J. H. A. Peck, M.D., Past President, K.M.S.

The Problem of Socialism—C. H. Benage, M.D., President-Elect, K.M.S.

The Medical Board—O. W. Davidson, M.D., Secretary, Board of Medical Registration and Examination.

The County Medical Society in Action—F. D. Taylor, M.D., Secretary, Clay County Medical Society.

The Kansas Medical Society and Current Legislation—F. R. Croson, M.D., President, K.M.S.

Following the day's program a social hour and banquet were held at the Continental Hotel for all juniors and seniors and their escorts. Mr. Bill Vaughan of the Kansas City Star was the banquet speaker. He presented numerous topics in a humorous manner and then gave a serious, sincere and timely discourse on "Socialism and its Undesirable Effects upon Medicine."

It is impossible to ascertain the amount of good these programs accomplish. Certainly the students love it all and their sincerity can well be judged by the numerous intelligent questions they ask. The men who have taken part in the programs during the past three years are very enthusiastic about this contact. It seems to bind the school and the individual students much closer to the Society and one cannot help but feel the mutual good will and fellowship which are ever present. Let us make these programs permanent and let us make them better and better.

I extend my personal thanks to everyone who appeared upon the program or who contributed in any way to make the day the success which it was.

Sincerely,

F. R. Croson M.D.

EDITORIAL COMMENT

Process of Evolution

In 1947 your Editorial Board established what at that time was hoped to be the first of an annual and permanent Kansas University issue of the Journal.

In reality, the first issue came about by chance. As we sat in the monthly Board meeting in February, we found that the only scientific papers we had were from Kansas University sources.

Now, in 1951, we are publishing this, our fifth annual issue.

At that meeting when the first issue was born, several things were discussed. First, that all factors pertaining to the science and art of the practice of medicine in this state or in any other state should revolve around the medical school. Second, that the scientific material that might be prepared from original and clinical research within the state should be published in our own Journal and not be scattered hither and yon, thereby helping to elevate Kansas medicine to its correct level in this country and abroad. Third, that it might be a definite stimulus to the young men and women who have availed themselves of the opportunity of studying medicine in our university, and at a great expense to our tax-paying citizens, realizing that Kansas medicine is progressive, to remain in our state and help carry this prospected goal nearer to the ideal. Fourth, that it would stimulate the profession of the state to better clinical observations and better medical writing because there would be more competition.

In 1949, when Oscar Davidson was our president, the first Kansas Medical Day was originated and held at the medical school in Kansas City. This is the one day in the year that the entire student body of the medical school gets together. It is particularly fitting that this day falls in March and coincides with the month when the Kansas University issue of the Journal is published. On this day the students are entertained by the Kansas Medical Society.

At the Kansas Medical Day in 1950, when Haddon Peck was our president, the Society was so impressed with the interest demonstrated by the student body that they established at the university a student loan fund of $10,000. The only stipulation to the management of this fund was that no one student could borrow more than $500.

During the past two or three years, under the leadership of Haddon Peck and Franklin Murphy, the Kansas Rural Health Plan was launched, and successfully so. There are many rural areas that have up to date medical clinics, built by the communities and manned by young physicians. These communities are now receiving medical care of a high plane without having to go outside those communities to get it.

Glen Shepherd of the university is one of our associate editors. It is through his efforts that this fifth K.U. issue of the Journal goes to press. He brings to our attention that the contributors from the university are now desirous of having their material printed in our Journal and are not looking elsewhere to have it printed.

And as we sit back and muse a little, we can hope at least that the first annual Kansas University issue of the Journal, born of necessity and by chance, may have played a very small part in the cementing of professional and individual relationships so that these and other worthwhile things have come to pass.

Doctors, Duty, and Dedication

Under the above caption Dr. Aldrich C. Crowe, president of the Medical Society of New Jersey, answered a physician who criticized action taken by the state's committee on physician resources. Although his opinions may not be shared by all members of the medical profession, his editorial provokes thought. A portion of the editorial is reprinted below.

"The constitutionality of Public Law 779 (the 'doctor-draft' law) is something we cannot adjudicate. So long as it remains on the statute books, it is the law of the land and we must obey it. Of course it *is* discriminatory. We doctors enjoy considerable discrimination in our favor—any honest physician can list these for himself. Are we to accept smugly all these perquisites but cry 'discrimination' when a cognate duty is imposed? We ask our unhappy correspondent just how *he* would obtain medical officers if too few volunteered. Would he recall those doctors who already had had foreign service, and whose commissions perforce had not yet expired? Or just let the soldiers go without medical attention? If he can think of a fairer way of obtaining the needed medical officers, we would welcome hearing about it.

"Must doctors' children be deprived of their fathers because the latter are physicians? Sometimes, yes. If a physician is enjoying the companionship of his children one evening and a call comes that there has been a wreck at the railway yards, he will have to abandon the children and go on duty. The

clerk across the street can continue to wallow in domesticity. If a man is unwilling to have his private life disrupted by duty, then he should never become a doctor. Ninety-four per cent of army and navy personnel are *not* physicians. They too have children. More than 96 per cent of those in combat are *not* physicians. Their children stand a good chance of permanently losing their fathers. Perhaps our correspondent can never understand this, but whoever takes the Hippocratic oath dedicates himself to public service. Just a sweet sentiment is it? Well, when the chips are down, it leaps into life as a tough reality. We expect a fireman to accept interruption of private life as part of a duty. Are doctors less courageous than firemen? At considerable cost, society educated the doctor. No matter how much tuition fee you paid, you got a bargain. Society made up the deficit. In return society expects you to be on call for emergencies. Such is the covenant we make when we become doctors.

"The fear that regimentation will lead to socialization is the strangest charge of the lot. We are faced with an emergency in which survival rather than victory is the immediate, urgent goal. We still have a free system of medical practice. If through this system we can furnish doctors so desperately needed, we have a strong argument in support of our system. But the people simply will not stand being deprived of medical care for troops if under our individualized system every physician can decide to stay home when the going gets tough. This is our chance to show that under a free system we can police our own profession."

Suicide

Although suicide would not likely be classified as a topic of more than casual interest to physicians, the prevention of that great tragedy does lie within the field of preventive medicine. A study of the subject was made recently by Dr. Carl L. Kline, a diplomate of the American Board of Psychiatry and Neurology, and was reported in the Wisconsin Medical Journal.

In presenting general facts Dr. Kline says that suicide stands ninth among the causes of death and accounts for more fatalities than the five most common communicable diseases, in spite of the fact that many actual suicides are not reported as such. More than half of all suicides occur in persons 45 years of age or older, and three times as many men as women destroy themselves. The suicide rate increases with advancing years, and beginning with adolescence each decade shows a steady and consistent rise.

A discussion of current misconceptions includes mention of the fact that one who commits suicide must be insane, which Dr. Kline has found is not the case. He has also found it untrue that suicide is restricted to depressed patients, that those who talk of taking their own lives never do so, that there is an increase in such deaths in periods of economic depression.

The author compares suicide to cancer and refers to it as a condition about which a great deal is known but the cause of which is still being sought. He believes that all physicians should be alert to factors in the personality of patients who are potentially suicidal so that psychiatric consultation may be instituted to prevent tragedy.

The largest group of patients who are potentially suicide victims is composed of persons who look and feel depressed and have additional symptoms such as sleeplessness, hypochrondriasis, feelings of guilt, feelings of unworthiness and failure, loss of weight, inability to work efficiently, inability to concentrate, memory difficulty or carelessness about appearance.

Next are those with an elevated mood, excess energy, overtalkativeness and playfulness, patients who may be on the upswing from a period of depression or in a mental state preceding such a period. Other groups include persons who show much dependence upon others, those who indulge excessively in oral gratifications, victims of chronic illness who display no will to live, patients with strong feelings of guilt and a need for punishment who insist on satisfying such needs through surgical procedures. In the latter group are "surgical shoppers" who, by means of unconscious mechanisms, develop symptoms which demand surgical intervention.

Suicide should be viewed as a reaction of a developmental nature rather than as an isolated incident in a lifetime, according to Dr. Kline. Generally speaking such patients have always had difficulty in dealing with hostile impulses and aggressivity, and resort to suicide when they turn these hostile, aggressive impulses upon themselves. The medical man and the psychiatrist, by working together, can rehabilitate many troubled patients and avert a significant number of tragedies.

Appointment and assignment of women physicians, dentists, and allied specialists to active duty as reserve officers has been authorized by the Department of the Army. They will be appointed to duty under regulations currently providing for the commissioning of male officers and will be given opportunities for clinical practice. With the exception of forward medical installations in combat zones, they will be eligible for service in every type of military medical facility.

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Case Report from the University of Kansas Medical Center

Severe Pulmonary Edema in an Ambulatory Patient

CLINICAL PATHOLOGICAL CONFERENCE

Edited by Glen R. Shepherd, M.D., and Mahlon H. Delp, M.D., from recordings of the conference participated in by the departments of medicine, pathology, roentgenology, and the junior and senior classes of medical students.

Case Presentation

J. R., a 49-year-old white male, was admitted to the Kansas University Medical Center on October 14, 1950, and expired the same day.

Chief complaints were "tightness of the chest, shortness of breath, and hemoptysis of three days duration. Shortness of breath was first noted three days previously. At that time he developed tightness of the chest and a productive cough of bright red blood. On the evening of October 13, 1950, the patient was taken to the KUMC emergency room where he was examined and was given penicillin intramuscularly. The following day he was seen in the outpatient clinic. Because of the progression of symptoms, he was admitted to the hospital.

Past history: His wife stated that he had been short of breath for approximately one year and that during recent months he had seemed to be losing weight. There was no history of rheumatic fever, growing pains, or other significant symptoms.

Family history: Father died at 83 of a stroke; mother died at 73 of pneumonia. There was no family history of diabetes, heart disease, tuberculosis, or carcinoma.

System review: No history of substernal pain could be elicited on admission. Ankle edema had not been noted. The gastrointestinal, genitourinary, and neurological systems were entirely negative. The patient drank "six to eight shots of whiskey daily" and three to four beers daily. There had been no recent weight loss.

Physical examination revealed a well developed, slightly obese white male of approximately 49 years of age who appeared acutely ill. The blood pressure was 135/80 in the left arm, temperature 103°F., pulse 120, respiration 26, height five feet four inches, weight 165 pounds. The pupils were round and equal, reacting to light and accommodation. The fundi revealed nothing unusual. Extraocular movements were normal. The ears, nose, and throat were essentially negative. There was no cervical adenopathy. The thyroid gland was not palpable. There were coarse, crackling rales throughout both lungs. The percussion note at the apices was dull, the vocal fremitus was increased bilaterally. The heart was not enlarged. There was systolic murmur at the apex. The P.M.I. was in the fifth interspace at the midclavicular line. The heart sounds were clear and regular. There was no axillary or inguinal lymphadenopathy. No abdominal masses could be palpated. The lower extremities and the neurological examinations were negative.

Laboratory examination: Urine was acid in reaction with specific gravity of 1.034, a faint trace of albumin, sugar negative, and microscopic negative. Complete blood count at noon revealed rbc., 4,000,000; wbc., 8,950; hemoglobin 77 per cent; polys 88, lymphocytes 10, monocytes two.

Complete blood count at 8:00 p.m. on the same day showed rbc., 5,020,000; wbc., 37,550; hemoglobin 84 per cent; polys 86, 76 filamented and 10 non-filamented; lymphocytes 13; monocytes one.

The blood N.P.N. was 63.5 mgm. per cent, sugar 160 mgm., chloride 68 milli-equivalents, CO_2 16.8 meq. Sputum culture was negative for pathogens.

Hospital course: Shortly after admission the patient seemed to improve slightly, respiration and pulse slowing on bed rest. Penicillin, 200,000 units every three hours, was started on admission. At 6:00 p.m. the patient complained of increasing dyspnea. Moist rales were heard throughout the lungs. Oxygen by nasal catheter was started and 0.6 mg. of purodigin was given. Dyspnea became progressively worse. Digitalization was completed at 9:00 p.m. Aminophylline and atropine seemed to relieve the respiratory distress and the chest cleared slightly. However, the patient expired suddenly at 9:49 p.m., death being preceded by a convulsion.

Dr. Delp: Are there any questions concerning this patient?

Question: Were there any bacteriological studies done other than sputum cultures that were negative for pathogens?

Dr. Stockard (Medicine Resident): There were no organisms found in nose and throat culture which was negative after 10 days.

Question: I would like to know more about the sputum. Did he have lots of sputum?

Dr. Stockard: We had an emesis basin by the side of his bed and it was filled twice with quantities of watery bloody sputum.

Dr. Dimond (Cardiovascular Section): (First EKG was taken at 2:30 p.m.) The limb lead RST segments are depressed. The diphasic, flattened inverted T waves are abnormal findings. Axis deviation is normal. There are no changes to suggest pulmonary infarction.

MEAT...and the ANABOLIC PROCESSES

Providing generous amounts of complete protein essential to the anabolic processes of the human organism, meat is an outstanding protein food for maintaining not only nitrogen equilibrium but also positive nitrogen balance in the patient.

Only in the past two decades has been established the full significance of adequate protein nutrition for supporting the anabolic processes of the organism in physiologic stress.[1] The healing of all types of wounds, repair of regenerating parenchymal organs, detoxification, maintenance of normal fluid balance between the various compartments of the body, growth of replacement tissue in extensive burns, rapid manufacture of antibodies, normal phagocytic response, upkeep of the erythrocyte mass and plasma protein, and support of the enzyme systems are but some of the physiologic processes dependent upon the state of protein nutrition in the patient.

Due to the almost complete absorption of the digestion products of meat protein and its excellent indispensable amino acid balance, the protein of meat participates efficiently in the synthesis of new tissue protein. On the other hand, studies in liver regeneration after partial hepatectomy have shown that incomplete proteins of vegetable origin, fed alone, do not increase the protein of the impaired liver any better than a diet containing no protein.[2]

The high content of biologically complete protein, however, is not the only reason for including liberal amounts of meat in the dietaries of patients requiring a high protein intake. Meat is also an important rich source of iron and valuable amounts of essential vitamins—thiamine, riboflavin, and niacin, and the newly discovered vitamin B_{12} which, among its several functions, promotes the most efficient utilization of protein.

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The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

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A distinct depression of the RST segment is present in the chest leads. The same marked depression occurs all the way across. This is just the opposite of what you would expect in a man who is having an acute myocardial infarction. It is suggestive of the type of findings you see in a subendocardial ischemia or subendocardial anoxia.

(Second EKG was taken at 6:30 p.m.) The patient had not been completely digitalized at this time. There are several things that have changed. We see extrasystoles now, ventricular in type. Also a big S wave has appeared in lead 1 which was not there previously, suggestive, but not diagnostic, of strain on the right ventricle. The same finding could be due to a change in heart position. The rate has increased from 120 to approximately 150-160. The RST segment depressions have increased considerably. I believe the QRS complexes are wider.

Dr. Tice (Roentgenology): These films were made on the day of admission. They are characterized by an extensive soft granular type of infiltration which seems to run out from the hilar areas on both sides. The apices are relatively clear. The heart is not large.

The differential diagnosis to my mind consisted first of decompensation. This was unlikely because of the character of the infiltration and because of the size of the heart. Secondly, the question of tuberculous pneumonia was raised in my mind. I discounted it because no primary lesion was apparent. Azotemia, associated with a high NPN sometimes gives a dense hilar infiltration. I finally settled on pneumonia of unknown etiology.

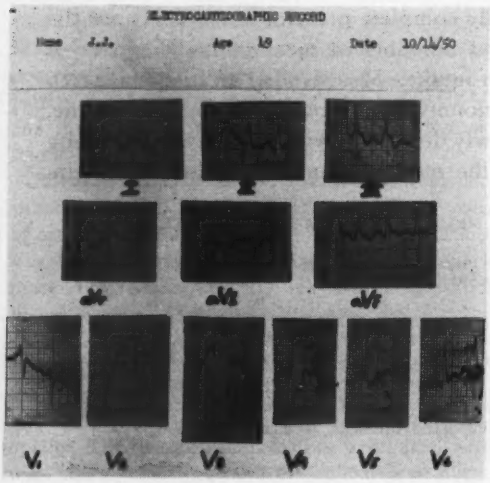

Figure 1. Interpretation: The significant findings are in the RST segment in leads 1 and 3 being reciprocally changed. These changes are very suggestive that the patient was having some cause for pulmonary hypertension with resultant right ventricular ischemia. This type of pattern is frequently seen in pulmonary embolism. There are frequent ventricular premature contractions present. Sinus tachycardia is the basic rhythm.

Differential Diagnosis

Mr. Dale Smith (Student:) When I saw this patient in the out-patient department, he was obviously acutely ill. He was dyspneic and was spitting up blood. With coughing, he produced a large amount of rather watery, frothy, and very bloody sputum. The history indicated the presence of chest pain from the first day of illness. He had chest pain during the day at work, continuing after he returned home that evening. That was when he started spitting up blood. On this basis, I thought of a pulmonary embolism.

The chief finding was hemoptysis. In a differential diagnosis, one has to consider all the causes of hemoptysis. The main causes considered are tuberculous pneumonia, mitral stenosis, pneumonia, lung abscess, pulmonary infarction, carcinoma either primary or secondary, bronchiectasis, as well as aortic or pulmonary aneurysm rupturing into the lung. There are other causes particularly higher in the respiratory tree such as in the larynx, but they don't produce this type of hemoptysis.

On an historical basis this man had been sick for three days and had hemoptysis for three days before he sought medical help. His stoical disposition creates the possibility of more severe chest pain than was supposed. There is a history of considerable alcohol consumption. Chronic alcoholism might predispose to pneumonia. The major physical findings are in the lungs, consisting of crackling rales, increased fremitus, and dullness to percussion.

We consider now the various factors in the differential diagnosis. Acute tuberculous pneumonia can be easily mistaken for pneumococcal pneumonia because both may present an acute onset of chills, prostration, aching chest, fever, rapid pulse, cyanosis, and hemoptysis. Other causes of pneumonia are influenza virus, primary atypical pneumonia, Friedlander's bacillus, and streptococcal pneumonia. Friedlander's bacillus causes about one per cent of pneumonias.

Bronchiectasis generally gives a characteristic history. However, an acute onset with hemoptysis could occur. Such a rapid course as this patient showed ruled out bronchiectasis.

Pulmonary infarction is a possibility, although there is no demonstrated site of origin for the embolus.

A lung abscess probably wouldn't present such an acute picture as this. A tumor of the lung, either primary or secondary, can be insidious initially and then have an acute onset with hemoptysis. There is no x-ray evidence of such lesion in the lungs.

I am left with two diagnoses: 1. A pulmonary infarct, even though there is no obvious source. 2. Pneumonia. Pneumococcal pneumonia does not usually give such a picture of hemoptysis. Primary

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atypical pneumonia is not as severe a disease. It is usually benign. It is rarely this virulent. Friedlander's bacillus pneumonia is frequently mistaken for pulmonary embolism.

I am going to say that he had a pneumonia, possibly Friedlander's. Otherwise, I think it was a pulmonary infarction.

Clinical Discussion

Dr. Delp: Dr. Allen, I am wondering what your explanation is for these pulmonary findings?

Dr. Max Allen (Medicine): A fatal pneumonia with a white blood count of 8,000 within 12 hours before death and with no organisms in the sputum seems like a very queer disease to me. I think if it is that, it must be a virus pneumonia. Pneumonia can be fulminating and even fatal, especially if imposed on pre-existing cardiovascular disease with associated pulmonary edema. Can we believe that blood count of 8,000 at noon on the day of death and 35,000 eight hours later? It is difficult to accept.

It might make me think of a virulent bacterial infection superimposed upon a less severe virus infection.

From some of the changes in the electrocardiogram and the description of chest discomfort it would appear that the heart was prominently involved in the terminal event and probably pulmonary edema existed at the time of death.

Dr. Delp: Dr. Tice, do you care to make any further suggestions?

Dr. Tice: I would like to go away out on a limb. I was impressed by this copious sputum. There is a condition known as pulmonary adenomatosis that is characterized by very copious sputum. I think the patient was ill for a longer period than the history indicates. This man was ill, according to his wife, since about the 10th of October. The condition raises the question as to whether it was a granulomatous or a malignant lesion.

Dr. Delp: I think Dr. Tice's point is well taken but whether or not it applies in this case we will learn later.

Dr. Peete (Medicine): I wonder if this patient really didn't have an overwhelming type of lobar pneumonia that was somewhat attenuated by penicillin; and the final event, the cause of death, nephritis.

Question: I would like to ask Dr. Dimond if there is any suggestion of a myocardial infarct from the changes in the electrocardiogram.

Dr. Dimond: Based on the appearance of the RST segment and the presence of the T wave, a myocardial infarction was not suggested.

Dr. Delp: In seeing this patient I was impressed by one feature. It was the most typical, classical pulmonary edema I have ever seen. It was a severe pulmonary edema, and simply because of the volume of the sputum it was confusing.

The next significant thing to be learned about the patient was the history of recurrent pain in the chest during the past year.

The next important feature is the history of episodic shortness of breath occurring in paroxysms almost every night. All of these should be important observations when coupled with electrocardiographic findings pointing to the heart as a principal focus for the disease state. I don't think we can say this man did not have a pulmonary disease in which toxemia was sufficiently great to have produced pulmonary edema. One other situation in which such massive pulmonary edema does occur is in the individual who has had a high grade mitral stenosis for a long period of time.

I feel that the primary disease was probably in the heart. And, obviously, we have one suggestion for painful types of heart disease. Now, may we hear what the pathologists found.

Pathology Report

Dr. Spicer (Pathology Resident): On general inspection the body was that of a well-nourished, obese, 49-year-old white male. The peritoneal cavity showed no fluid nor other positive findings.

The thoracic cavity contained 100 cc. of clear straw colored fluid in the right pleural space and 200 cc. of clear straw colored fluid in the left pleural space. The pleural surfaces were smooth, moist and glistening. There were adhesions at the bases and the organs had a normal relationship.

The pericardial sac contained 25 cc. of clear fluid and the surfaces were smooth, moist, and glistening. There were no adhesions.

The heart weighed 400 grams, was conical in shape. There were a few areas of softening involving the lateral wall of the left verticle and the apex. The tricuspid valve measured 14 cm., the pulmonary eight cm., the mitral 11 cm., and the aortic valve 8.5 cm. Both coronary arteries showed atheromata about one cm. from the ostia. There was a rather large area of necrosis under the apex of the heart involving the lateral wall of the left ventricle at the septum. The left ventricle measured 18 mm. in thickness and the right ventricle five mm. The heart appeared to be definitely dilated.

The right lung weighed 800 grams and the left lung weighed 600 grams. There were some adhesions at both bases bilaterally. The lungs were spongy in consistency. They showed considerable edema and exuded a large amount of fluid on inspection. There was congestion throughout both the lungs and a mucosanguineous exudate in the bronchi. The vessels appeared to be patent and normal.

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There were some petechial hemorrhages in the stomach and about 150 cc. of greenish fluid. The rugae were not distinct and the mucosa was intact. There were some petechial hemorrhages throughout the duodenum.

The provisional gross anatomical diagnosis: coronary atheromata, large apical and lateral myocardial infarction, cardiac dilatation, chronic passive congestion of the lungs and liver.

Dr. Wyatt (Pathology Resident): Of course the main findings are in the heart. The lung, however, is affected on microscopic examination. There was pulmonary edema present and much early passive congestion also. Heart failure cells in the alveoli were of giant proportions. This suggests repeated bouts of pulmonary difficulty because of the large number of heart failure cells. There were also a few clusters of smaller leukocytes which were mostly polys. This was interpreted as being an early hypostatic bronchial pneumonia. It was not extensive.

Particularly striking were the degenerative myocardial muscle fibers. These lesions were present for a month or longer. There was considerable fragmentation throughout the cardiac musculature as well as an organizing area of infarction.

There was considerable endocardial reaction microscopically which was not so evident grossly. This is the same type of reaction as seen through the myocardium. There was considerable fibrosis, and particularly striking were the number and types of histiocytic cells. This lesion extended down into the myocardium, forming somewhat of a syncytial appearance and replacing myocardial fibers.

Some of the lesions were probably of two to three weeks duration as shown by organization with numerous capillaries.

The cause of death and the events in which they occurred would be gross coronary occlusion with organizing myocardial infarction of the left anterior ventricle. Parenchymatous myocarditis, pulmonary edema, and passive congestion with acute dilatation of the heart were present. This heart also showed changes indicating possibly an earlier lesion.

Dr. Wahl (Pathology): It is obvious that this patient died from cardiac failure with a very severe pulmonary edema.

Dr. Dimond: I should like to ask the pathologist whether the infarct ran through the muscle. Was it from epicardium to endocardium? What surface was affected?

Dr. Wahl: It affected the endocardium more than the epicardium.

Dr. Dimond: The electrocardiogram indicated that the pathology was confined to the endocardium. It is said that 85 per cent of primary infarctions can be shown by EKGs. And that percentage is increased by taking leads at different areas across the chest. The accuracy of the electrocardiogram is directly proportional to the experience of the interpreter.

Regarding the care of pulmonary edema, one must first agree on the cause of pulmonary edema. Bedside clinicians for years have maintained that pulmonary edema is the same thing as left ventricular failure or cardiac asthma. And by saying left ventricular weakness or failure they infer that the man has pulmonary edema because the left heart has been unable to empty the lungs, the capillaries are therefore under an increased pressure and there is transudation of fluid with resultant edema.

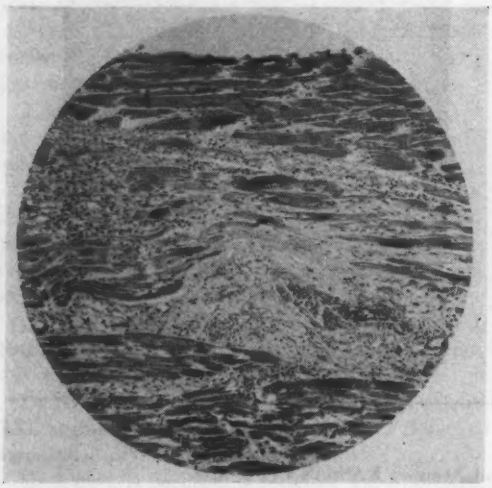

Figure 2-A. A section of myocardium showing fibrosis, perivascular inflammatory reaction, and diffuse inflammatory infiltrate of polys, lymphs, and histiocytes. x 60.

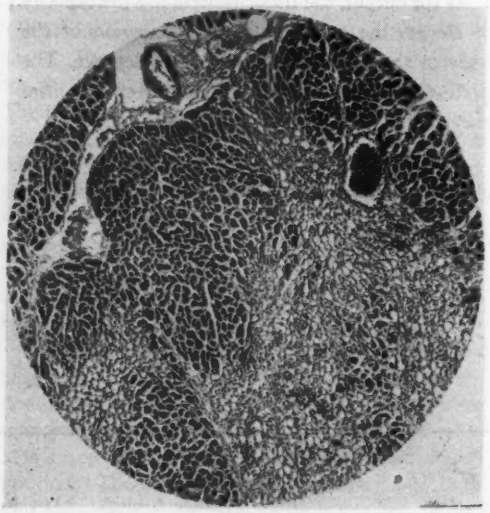

Figure 2-B. Degeneration and necrosis of myocardium is evidenced by vacuolated fibrils. Also numerous inflammatory cells including foamy histiocytes are present. x 30.

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Physiologists for years have maintained that this concept is not true and that there is no experimental evidence to prove the point. They say that pulmonary edema is primarily cerebral and neurogenic in origin, perhaps associated with cerebral anoxia. They cite as evidence that a person can have an infarct in the right ventricle and develop pulmonary edema with no change in the emptying time of his left ventricle. Further evidence is the fact that the best treatment for pulmonary edema is morphine which has a cerebral effect and no myocardial effect. I personally agree with this thought—that pulmonary edema is neurogenic, reflex in origin, and controlled by higher nerve centers.

The best treatment for pulmonary edema with that thought in mind then is: 1. Give morphine. Morphine has a central sedative action. 2. Give oxygen to correct anoxia. Give the oxygen under positive pressure so that there is a counteracting pressure to the increased intercapillary pulmonary pressure. That is a very dramatic way of treating pulmonary edema. It is a specific form of treatment.

The effect of aminophylline is beneficial if given slowly. If aminophylline is given rapidly it increases the cardiac work. If given slowly, it is a bronchial dilator, and in treatment of pulmonary edema there is marked bronchial spasm accentuating the anoxia. Slowly given intravenous aminophylline, 3¾ to 7½ grains, is a specific treatment. Atropine may be beneficial as a drying agent and perhaps in blocking reflex vagal effect. As to the true usefulness of atropine, there is disagreement. However, if it is to be used, it should be given in large amounts of 1/50 to 1/75 of a grain.

In summary, I think the treatment of pulmonary edema should be: 1. Good big repeated doses of morphine. 2. Oxygen under pressure. 3. Aminophylline by vein. 4. If you think the patient has cardiac failure due to intrinsic heart disease, digitalize him and digitalize him rapidly. And when you wish to use tourniquets or to bleed him, I suspect that you are doing something while waiting for other drugs to take effect. 5. Atropine may be helpful, if an adequate amount is given.

Dr. Major (Medicine): It is difficult for me to dismiss the possibility of pneumonia in the face of fever of 103°F. and a white blood count of 37,000.

Dr. Wahl: I looked over many blocks of tissue taken throughout the lungs. We were looking for pneumonia because of the history. We distinctly tried to find evidence of pneumonia. There were just a few polys in any alveolus. No single space had more than five or six polys. I knew this count went up to 37,000 but he certainly hasn't microscopic evidence of pneumonitis.

Summary

The phlegmatic individual presenting himself as an ambulatory patient may easily diminish the physician's sharpness and acuity in recognition of serious illness. Just such a situation existed here. A further lesson is to be learned regarding the importance of historical omissions. Failure to learn of the presence, duration, and character of chest pain resulted in preliminary ignorance of the probable presence of coronary artery disease. Pulmonary edema is easily most often seen by the physician in the home in situations so emergent that the patient cannot be moved. Nevertheless the physical characteristics of pulmonary edema are so clear that it should never be confused. Its presence should indicate the most likely site of disease to be in the heart.

Finally, there is increasing evidence of central and neurogenic factors in its origin.

Fifth Medical School Issue

An invitation to contribute scientific papers to this, the fifth annual University of Kansas School of Medicine issue of the Journal, was extended to the different departments at the school, and the response was immediate and generous. Since space limitations prohibit use of all the material in one issue, several papers were scheduled for publication at different times.

Two papers which appeared in the February issue, "The Problem of Success and Failure in the School Age Child" by Dr. Rodger A. Moon and "Rapid Rehabilitation of Disabilities of the Knee" by Dr. Donald L. Rose and Lilyan G. Warner, were submitted for the university issue.

The following papers will be published in later issues of the Journal: "The Surgical Treatment of Extensive Skin Cancer of the Lower Eyelid" by Dr. Creighton A. Hardin and Dr. David W. Robinson, "Infantile Cortical Hyperostoses" by Dr. John F. Bowser, "The Diagnosis and Treatment of Subdural Hematomas in Infants" by Dr. John R. Patterson and "A Roentgenographic Study of Edentulous Jaws" by Dr. Ralph W. Edwards.

Nursing needs have increased in spite of lower death rates, longer life expectancies, and a generally healthier population. The number of people in the United States has increased by an estimated 16,000,000 since the 1940 census. More people are now living to an older age than ever before, and consequently the diseases and disabilities of older people have multiplied. A high standard of living has prevailed since the early years of the war, medical prepayment plans have spread, and public health services have been expanded in many areas. In 1940, 10,087,000 patients were admitted to hospitals in the United States, but by 1948 the number of hospital admissions had risen to 16,422,000.—*Public Health Reports, August 5, 1949.*

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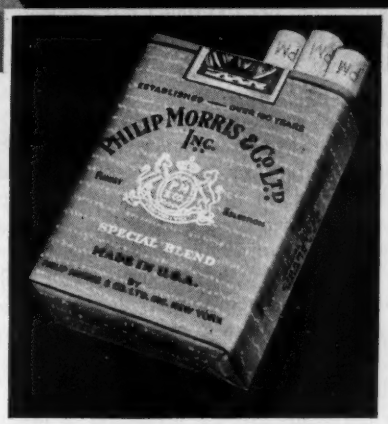

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**Proc. Soc. Exp. Biol. and Med., 1934, 32, 241-245; N. Y. State Journ. Med., Vol. 35, 6-1-35, No. 11, 590-592; Laryngoscope, Feb. 1935, Vol. XLV, No. 2, 149-154; Laryngoscope, Jan. 1937, Vol. XLVII, No. 1, 58-60*

BLUE SHIELD

Blue Cross Experience Affects Blue Shield

A great deal has been achieved in establishing a better understanding of Blue Shield among participating physicians. It has been done primarily through the Blue Shield Relations Committee of the Kansas Medical Society. Up until now very little has been done in the way of bringing to the attention of doctors information on the workings of Blue Cross.

Both Blue Cross and Blue Shield are offered to the public as a single package program. During the past year Blue Shield enrollment has been slightly ahead of Blue Cross in that many people who were previously members of Blue Cross are now adding Blue Shield to their coverage. The present membership in Blue Cross is 410,046 members, whereas Blue Shield has 258,589. It is foreseen that eventually Blue Shield enrollment will come pretty close to equalling the enrollment in Blue Cross. However, since both plans are sold as a package the cost of Blue Cross membership has a direct bearing on the acceptance of Blue Shield by the public and vice versa. The public thinks of both plans as a single organization.

During 1950 Blue Cross paid out in terms of hospital service for members slightly over 90 per cent of its income. Only 10 per cent of the income was used for administrative expenses. The number of hospital admissions per 100 Blue Cross members increased during the year. The rate of admissions during 1948 was 14.2 members per 100, while this rate for 1950 was 16.2 members per 100. This means that Blue Cross experienced a rate of over 8,000 additional admissions per year. Furthermore, the total hospital charges per case increased 24 per cent during this period. In 1948 the average charge per case was $73.32 whereas the average charge per case in the last quarter of 1950 was $91.43. This experience means that it will be necessary for Blue Cross to work out a new contract during 1951. It is expected that this new contract will involve an increase in dues as well as an increase in benefits.

This information is presented in the interest of showing physicians that the utilization of hospital service by Blue Cross members has a direct bearing on the over-all Blue Cross-Blue Shield program. At a recent Hospital-Physician Relations Conference of Blue Cross-Blue Shield Plans in Chicago a great deal of emphasis was placed upon the need for both organizations to consciously plan a program of physician relations to inform physicians on Blue Cross problems. Doctors are the final arbiters as to whether or not Blue Cross members shall go to the hospital and as to what services the members shall receive in the way of drugs, laboratory, etc. If the individual physician is aware of the effect which unnecessary use of service will have upon the Blue Cross-Blue Shield future, it is felt that he will help to curb some of the demands of patients for unnecessary hospital admission and service.

The Kansas Hospital Association is studying the over-all problem of high use by members. It is expected that the association will make recommendations to the effect that Blue Cross bring the proper information on this question to the doctors of the state. Actually a group of physicians already exists for helping in this program. The nucleus of this group might well be the eight physicians serving on the Blue Cross Board of Trustees. Since doctors have a voice in determining Blue Cross policy it is felt that they will wish to assume reasonable responsibility in keeping the plan in a position where it can provide the greatest possible service at the lowest possible cost. The entire information program to eliminate unnecessary use of service will be on a voluntary basis. It is simply a question of asking physicians to "turn out the lights in rooms that are not being used." In other words, all that Blue Cross will ask is that *the true needs of the patient as interpreted by the physician* be the deciding factor as to whether a patient is to receive service under his Blue Cross membership.

Some startling savings might result through wholehearted cooperation by physicians. For example, if one-half day could be cut off the average length of stay, it would produce a savings of over $270,000 per year. Or if the rate of admission could be cut from 16.2 admissions per 100 members to 15.2 per 100, it would produce a saving of 4,000 cases or over a quarter of a million dollars. All such savings would mean a better package program at less cost, which in turn should mean a more rapid rate of enrollment.

Discuss Field Medical Equipment

More than 100 medical and dental officers of the United States Army, Navy and Air Force met in Montreal recently with representatives of the United Kingdom and Canada to discuss field medical and dental equipment for the services of the three countries. Among the subjects discussed were medical equipment in the Arctic, Arctic survival, pharmaceutical supply units, field surgical units, field dental units, field malaria and epidemic disease control, field radiographic and laboratory work, antiseptics, and Arctic clothing at sea.

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Hamblen, E. C.: North Carolina M. J. 7:533 (Oct.) 1946.

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*Perloff, W. H.: Am. J. Obst. & Gynec. 58:684 (Oct.) 1949.

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92nd Annual Meeting of K.M.S.

Committees of the Shawnee County Medical Society are now completing arrangements for the 92nd annual meeting of the Kansas Medical Society, to be held in Topeka, May 14-17, 1951. A number of well known scientific speakers from out of the state are scheduled to present papers of interest to specialists and general practitioners alike, and sectional meetings which will feature Kansas physicians as speakers are being arranged.

Among the medical specialists who will present the scientific program are the following:

Ethan A. Brown, M.D., lecturer in allergy, Tufts College Medical School, Boston.

Charles H. Burnett, M.D., professor of internal medicine, Southwestern Medical School of the University of Texas, Dallas.

Tullos O. Coston, M.D., assistant professor of ophthalmology, University of Oklahoma School of Medicine.

Stuart C. Cullen, M.D., chairman, Division of Anesthesiology, State University of Iowa, Iowa City.

William J. Engel, M.D., urologist, Cleveland Clinic, Cleveland, Ohio.

Thomas F. Frawley, M.D., internist, Peter Bent Brigham Hospital, Boston.

N. Frederick Hicken, M.D., associate clinical professor of surgery, University of Utah School of Medicine.

Robert L. Jackson, M.D., associate professor of pediatrics, State University of Iowa.

John H. Moore, M.D., obstetrician and gynecologist, Grand Forks Clinic, Grand Forks, North Dakota.

Clyde L. Randall, M.D., professor of obstetrics and gynecology, University of Buffalo School of Medicine, Buffalo, New York.

John R. Schenken, M.D., professor of pathology and bacteriology, University of Nebraska, Lincoln.

Albert C. Stutsman, M.D., instructor in clinical otolaryngology, Washington University School of Medicine.

Round table luncheons will be held each day of the meeting, and the usual entertainment features, highlighted by the annual banquet on May 16, will be provided.

Hotel reservations should be made immediately Those who plan to attend should write directly to the hotel of their choice. The larger hotels in Topeka are the Jayhawk and the Kansan.

Nominations for Society Offices

The House of Delegates of the Kansas Medical Society at the annual meeting in 1950 amended the constitution and by-laws of the Society to provide for a nominating committee to prepare a slate of candidates for Society offices. The amendment also provided that at least two nominations must be made for each elective office and that the names of the candidates be published in the Journal. Additional nominations may be made from the floor.

A nominating committee of five members, selected from a list of past presidents of the Society, was elected by the House of Delegates. The committee met in Topeka last month, with all members attending, and prepared the following slate from which officers will be elected at the annual session in Topeka on May 17, 1951: president-elect, Dr. W. F. Bernstorf, Winfield, and Dr. Barrett A. Nelson, Manhattan; first vice president, Dr. J. V. Van Cleve, Wichita, and Dr. Lucien R. Pyle, Topeka; second vice president, Dr. Conrad M. Barnes, Seneca, and Dr. Murray C. Eddy, Hays; treasurer, Dr. Vernon Wiksten, Topeka, and Dr. Byron J. Ashley, Topeka; secretary, Dr. Dale D. Vermillion, Goodland, and Dr. Robert Sohlberg, McPherson; delegate to A.M.A., Dr. John M. Porter, Concordia, and Dr. Thomas P. Butcher, Emporia; alternate, Dr. J. L. Lattimore, Topeka, and Dr. P. E. Hiebert, Kansas City.

DEATH NOTICES

JOHN GURLEY MISSILDINE, M.D.

Dr. J. G. Missildine, 66, an active member of the Sedgwick County Medical Society, died at his home in Wichita, January 30. He was graduated from the University of Maryland School of Medicine and the College of Physicians and Surgeons, Baltimore, in 1911 and received his Kansas license in 1912. He went to Wichita from Parsons in 1920, and continued to practice there except for a time during World War II when he served in the Army medical corps. He specialized in urology and dermatology.

* * *

JONATHAN BASIL CARTER, M.D.

Dr. J. B. Carter, 73, an active member of the Central Kansas Medical Society, died February 3 at Ellsworth. After his graduation from the University of Kansas School of Medicine in 1910, he began practice in Lorraine and later moved to Wilson. He served in the medical corps in both world wars and remained in the armed forces after World War II, retiring several months ago. Dr. Carter was interested in civic and political matters. He served Wilson as its mayor for two terms and later represented the 32nd district in the state senate.

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		First 3 Months for Hospital Disability.
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$	5,000	Cash, & $400 monthly first 2 years, $300 monthly thereafter.
		Adjusted benefits for disabilities occurring after age 60.

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ACTIVITIES OF MEMBERS

The American College of Anesthesiologists announces that Dr. William O. Martin, Topeka, and Dr. Edgar M. Sutton, Salina, became fellows of the college recently.

* * *

Dr. C. M. Fitzpatrick, Salina, has been appointed health officer of Saline County, filling the vacancy created by the resignation of Dr. L. S. Nelson, Jr.

* * *

Dr. Richard L. Sutton and Richard L. Sutton, Jr., Kansas City, were recently elected to corresponding membership in the Swedish Dermatological Society.

* * *

Dr. J. W. Cheney and Dr. Paul C. Carson, Wichita, were honored by the Kansas Children's Service League last month in recognition of 25 years of service to the organization.

* * *

Dr. A. J. Randolph, Kansas City, was named deputy coroner for Wyandotte County recently.

* * *

Dr. F. C. Beelman of Topeka, deputy of health for the Kansas civil defense program, discussed the atom bomb before the Lions Club at Perry last month.

* * *

Dr. R. J. Metcalf, El Dorado, has been named acting director of the Butler-Greenwood-El Dorado board of health, taking the place of Dr. Richard Reece, who is to report for active duty with the Army on March 16.

* * *

Dr. C. T. Sills, Newton, gave a history of the fight against tuberculosis at a meeting in Newton preceding the county chest x-ray survey.

* * *

Dr. Charles R. Svoboda, Chapman, has been appointed health officer for Dickinson County for 1951.

* * *

Dr. M. O. Steffen, Great Bend, stated objections to socialized medicine at a meeting of the A.A.U.W. in Great Bend last month.

* * *

Dr. J. L. Wentworth, Dr. E. H. Clayton and Dr. W. G. Weston, Arkansas City, presented hospital equipment valued at $1,800 to the new Memorial Hospital there.

* * *

Dr. John B. Nanninga, Newton, has been named chief of health services of the Newton-Harvey County civil defense group.

Dr. J. D. Colt, Sr., Manhattan, was the subject of a feature story in a recent issue of the Manhattan Tribune. The story told of his many professional and civic activities and mentioned that he was president of the Kansas Medical Society in 1933.

* * *

Dr. S. A. Scimeca, Caney, has announced that he will practice in Elk City on Wednesday of each week.

* * *

Dr. R. E. Capsey, Centralia, has been named health officer for Nemaha County.

* * *

Dr. Henry C. Eichelmann has been named civil defense director for the city of Norwich.

* * *

Dr. Donald P. Trees, Wichita, discussed "What's New in Medicine" at a meeting of the Hyde P.T.A. in Wichita recently.

* * *

Dr. A. R. Chambers, Iola, is serving as physician for the Durite Corporation, manufacturers of lawn mowers, in Iola.

* * *

Dr. G. I. Thacher, Waterville, was recently named health officer of Marshall County, replacing Dr. C. M. Newman, Axtell, who has retired.

* * *

Dr. E. J. Ryan, Emporia, spoke on "World Health Problems" at a recent meeting of the Emporia branch of the A.A.U.W.

* * *

Dr. J. H. Johnson is serving as chairman of the health group of the El Dorado Red Cross Disaster Committee.

* * *

Dr. Edward H. Johnson has closed his office in the State Bank building in Peabody and has not announced plans for future practice.

* * *

Dr. James A. Wheeler, Newton, discussed infection before a group of nurses in District Four at a meeting held in Newton recently.

* * *

Dr. Arthur E. O'Donnell, Dr. Fred W. O'Donnell and Dr. Harry E. O'Donnell, Junction City, recently purchased new office equipment and gave old machines to the Junction City high school for observation work. The gift included three x-ray machines, two fluoroscopes, a developing tank and a vibrator.

* * *

Dr. G. L. Campbell is representing the medical profession on the Arkansas City civil defense board.

* * *

Dr. D. Cramer Reed, Wichita, addressed 150 volunteer workers for the American Cancer Society at

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OF
WATER
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a meeting held last month in Wichita. He spoke on "The Cancer Problem Today."

* * *

Dr. and Mrs. F. A. Garvin, Augusta, recently observed their 58th wedding anniversary.

* * *

Dr. Robert L. Stevens, Garnett, reported to the Army at Camp Carson, Colorado, on February 5, to serve as a first lieutenant in the medical corps. His wife, Dr. Mildred Julius Stevens, will continue practice in Garnett.

* * *

Dr. Ralph G. Ball represents the medical profession in the civil defense organization for Manhattan and Riley County.

* * *

Dr. T. D. Ewing, Bushton, spoke on "Occupation of Japan" before the Lions Club at Holyrood recently.

* * *

Dr. R. R. Snook, McLouth, was one of the speakers at a meeting of the Jefferson County Cancer Society at Valley Falls recently.

* * *

Dr. Edward D. Greenwood, director of the Southard School, Topeka, was one of the speakers at the organization meeting of the Kansas Hospital Recreation Society held at Topeka, January 27. Members of the group are recreational therapists at nine Kansas institutions.

* * *

Dr. C. W. Wilson, St. Francis, was named Cheyenne County physician recently.

* * *

Dr. Charles S. Davis, who has been practicing in Galena for 17 months, received orders to report for active duty in the Army early in February. He has been commissioned a first lieutenant.

* * *

Dr. Chester H. Smith has been named to direct matters affecting health on the civil defense council at Pittsburg.

* * *

Dr. George S. Hopkins, surgeon at the Santa Fe Hospital, Topeka, was recently elected to fellowship in the American College of Surgeons.

* * *

Dr. W. T. Read has been named director of health for the Coffeyville civil defense program.

* * *

Dr. Gertrude E. Martin of Sterling, who practiced a few years ago at the Trueheart Clinic, opened an office for private practice in the Culbertson Building there on February 20.

* * *

Dr. Robert G. Smith, Topeka, was recently elected to fellowship in the International College of Surgeons.

* * *

Dr. B. E. Miller, Council Grove, announces that Dr. Byron A. Yost and Dr. Robert W. Blackburn are now associated with him in practice. Both are graduates of the University of Kansas School of Medicine and have been practicing in Wellington.

* * *

Dr. Lee H. Leger, Kansas City, took part in a radio program on February 11 over station WDAF, Kansas City. The program was sponsored by the Kansas Heart Association, of which he is vice president.

* * *

Dr. B. H. Mayer, Ellsworth, is chairman of the public health committee for the Ellsworth County civil defense unit.

* * *

Dr. F. E. Harvey, who has practiced in Minneapolis for 46 years, was the subject of a feature story in the Minneapolis Messenger on February 15.

* * *

Dr. James Pretz, Kansas City, has resigned as physician for Wyandotte County and is to report for active duty in the Army on March 12.

* * *

Dr. O. C. McCandless, who has been Marion County health officer for nine years, resigned that position to become director of the city-county health unit in Cheyenne, Wyoming. He began his new work March 1.

* * *

Dr. Raymond J. Beal, Fredonia, addressed the Business and Professional Women's Club in that city at its February meeting, speaking on the subject of cancer.

* * *

Dr. B. V. Thompson, who has been associated with the Hatcher Clinic at Wellington for the past five years, left the organization March 1 and went to Florida for several weeks visit. He will maintain his home in Wellington but has not announced plans for the future because of possible entry into the military forces.

Objectives of the Commission on Chronic Illness are listed as follows: (1) modify society's attitude that chronic illness is hopeless; (2) define the problems arising from such illness in all age groups; (3) coordinate separate programs for specific disease with a general program designed to meet the needs of all the chronically ill; (4) clarify the relationships of all groups working in the field; (5) stimulate in every locality a well-rounded plan for the prevention and control of chronic disease and the care and rehabilitation of the chronically ill.

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Karl J. Waggener, M.D. **Wendell T. Wingett, M.D.**

COUNTY SOCIETIES

A meeting of the Shawnee County Society was held at Topeka, February 5. Dr. Roger W. Ridley, Rochester, Minnesota, spoke on "Application of Regional Anesthesia in Diagnosis and Treatment." At the business session four Topeka physicians were elected to membership, Dr. Richard L. Merkel, Dr. M. D. Morris, Dr. Donald R. Pierce and Dr. William H. Wood. For the benefit of physicians from outside the county who attend meetings in Topeka, the society announces that its meeting date has been changed to the Tuesday following the first Monday of each month.

* * *

Dr. V. J. Vaughn, Axtell, was named president of the Marshall County Society at a recent meeting held at Marysville. Dr. D. M. Diefendorf, Waterville, was elected vice president and Dr. R. D. Hughes, Frankfort, was named secretary.

* * *

The annual dinner meeting of the Cowley County Society was held recently at the Esquire Club south of Arkansas City. Wives of the members were guests.

* * *

New officers of the Nemaha County Society were elected at a meeting held last month in the office of Dr. C. M. Barnes, Seneca. Dr. Arthur H. Haynes, Sabetha, is president; Dr. C. C. Hunnicutt, Sabetha, vice president; Dr. John Howard Gilbert, Seneca, secretary-treasurer. After the business session the group joined members of the Auxiliary for a social hour at the Barnes home.

* * *

Physicians of Chautauqua and neighboring counties were guests of the Sedan Chamber of Commerce at a dinner at the school cafeteria on February 1. The group was shown through the new Sedan City Hospital, which held its formal opening on the following Sunday.

* * *

Dr. Walter F. Kvale, internist from the Mayo Clinic, Rochester, Minnesota, was speaker at the February meeting of the Sedgwick County Society. His subject was "Occlusive Arterial Diseases."

* * *

A meeting of the Marion County Society was held at Coons' Restaurant, Marion, on February 7. Dr. H. F. Janzen, Hillsboro, and Dr. T. C. Ensey, Marion, presented the scientific program.

* * *

The Rice County Society elected officers at a meeting held at Lyons in January. Dr. T. D. Ewing was named president; Dr. J. C. Dysart, vice president; Dr. E. R. Hill, secretary-treasurer. A professional film, "Hemi-colectomy," was shown.

* * *

A meeting of the Douglas County Society was held February 13 at Watkins Hospital, Lawrence. Dr. Ralph I. Canuteson spoke on the use of BCG vaccine in tuberculosis control.

* * *

The Crawford County Society met at the Hotel Besse, Pittsburg, February 22 with 28 members attending. Dr. Marvin F. Hall, Joplin, Missouri, spoke on opaque media in x-ray diagnosis. At the business session the group endorsed the efforts of the Southeast Kansas Tuberculosis Association to obtain a tuberculosis sanitarium for that section of the state. The society also voted to ask for extension courses for 1951 and 1952 with Mt. Carmel Hospital offering its facilities.

Proposed Change for K.M.A.S.

A change in the constitution of the Kansas Medical Assistants Society, to provide for a nominating committee, has been proposed and will be voted on at the annual meeting of the organization in May. The wording of the proposed amendment is as follows:

The annual nomination for president, president-elect, vice president, secretary and treasurer shall be made by a nominating committee to be appointed by the president at least six (6) months prior to the annual election. The nominating committee shall be composed of five members and shall be appointed by the president to represent the Society on a geographic basis. The chairman of the nominating committee shall be a past president of the Society.

Two names shall be presented by the nominating committee for each office, no nominations to be made without the consent of the nominee. Nominations for each office may also be made from the floor.

The candidate receiving the greatest number of votes shall be elected.

A resolution passed by the Montana Chamber of Commerce recently reads as follows: "We oppose federal medicine for the simple reason that like every other key plank to Marxian socialist doctrine it simply will not work. No nation in the world has better medical care or is in better health than the United States. This obvious fact leads us to believe that socialized medicine is a scheme to exploit the doctors who are a minority in order to pretend to distribute benefits to the voting majority who are the people. This is dishonest."

Storm Warning

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Medical Director

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ANNOUNCEMENTS

The Mid-Continent Psychiatric Association, a branch of the American Psychiatric Association, will meet March 31 and April 1 at the Hotel President, Kansas City, Missouri. Among the speakers will be Dr. Charles F. Von Salzen of Hartford, Connecticut, Dr. Martin H. Hoffman of Detroit, Michigan, and Dr. Guy F. Witt of Dallas, Texas. Physicians are cordially invited to attend. Those planning to be present should notify Dr. Paul Hines, 2625 West Paseo, Kansas City, Missouri.

* * *

The Michael Reese Hospital Postgraduate School, Chicago, announces three spring courses. First is a course on clinical dermatology, April 2 to April 7, with clinics and lectures conducted by members of the Department of Dermatology and Syphilology. The second course is "Surgery—Indications, Pre- and Post-Operative Care," April 9 to April 14. "Recent Advances in Internal Medicine" is the subject of the third course, April 30 to May 12.

Complete information may be secured from Dr. Samuel Soskin, Dean, 29th Street and Ellis Avenue, Chicago 16, Illinois.

* * *

The 1951 meeting of the American Goiter Association will be held in the Deshler-Wallick Hotel, Columbus, Ohio, May 24, 25 and 26. The program will consist of papers dealing with goiter and other diseases of the thyroid gland, dry clinics and demonstrations.

* * *

The fifth annual Symposium on Fundamental Cancer Research of the University of Texas M.D. Anderson Hospital for Cancer Research will be held at the Texas Medical Center, Houston, April 20 and 21. A meeting of the South Central Region of the College of American Pathologists will also be held. Dr. Fred W. Stewart will deliver the second Bertner Foundation Lecture at the banquet on April 21. Further information may be obtained from William O. Russell, M.D., 2310 Baldwin Street, Houston, Texas.

* * *

The Kansas City Southwest Clinical Society again this year is offering awards for papers by graduates in medicine serving residencies or internships in approved hospitals in this area, Arkansas, Colorado, Iowa, Kansas, Missouri, Nebraska, or Oklahoma. Each paper must represent original work, a review of clinical cases in the hospital or actual experimental work in the laboratory in which the contestant is serving his residency or internship. The winner will receive $500 and will present his thesis before the general assembly at the fall clinical conference of the society. Winners of second and third awards will receive $100 and $50, respectively.

Applications, endorsed by the chairman of the resident-intern committee or the superintendent of the hospital, must be made in writing and mailed to the society by April 15, 1951. Papers will be accepted to August 2, 1951. The address of the society is 630 Shukert Building, Kansas City 6, Missouri.

* * *

A course in ophthalmology and otolaryngology will be offered at the University of Kansas Medical Center, April 16-20. The list of guest instructors includes Dr. F. Bruce Fralick, professor of ophthalmology and chairman of the department, University of Michigan School of Medicine; Dr. Howard I. House, clinical professor of otolaryngology and head of the department, University of Southern California School of Medicine; Dr. Carl C. Johnson, associate surgeon in ophthalmology, Harvard Medical School and the Massachusetts Eye and Ear Infirmary, Dr. Irving H. Leopold, attending ophthalmologist, Wills Eye Hospital, and consulting ophthalmologist, University of Pennsylvania Medical School; Dr. Theodore E. Walsh, professor of otolaryngology and chairman of the department, Washington University School of Medicine.

* * *

A course in the theory and techniques of autoradiography will be held during the coming summer by the Special Training Division of the Oak Ridge Institute of Nuclear Studies, beginning on July 2 and continuing for from three to four weeks. Subjects to be covered in lectures and laboratory sessions will include the theory of the photographic process, reaction of ionizing particles with photographic emulsions and the interpretation of results; techniques of making gross and microscopic autoradiograms. Registration for the course will be $25. Application forms may be secured from Ralph T. Overman, chairman, Special Training Division, Oak Ridge Institute of Nuclear Studies, P. O. Box 117, Oak Ridge, Tennessee.

The first 4,000 replies received to a questionnaire sent out by a Texas Congressman, Representative Ben Guill, indicate that Texans do not favor socialized medicine. The question was asked as follows: "Do you favor the proposed national health insurance law which would provide medical and hospital care for everyone (also known as Socialized Medicine) at a cost of an estimated six billion dollars in new taxes?" One hundred sixty-seven answered yes, 3,623 replied in the negative, and 115 expressed no opinion.

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$25.00 weekly indemnity, accident and sickness	Quarterly
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$75.00 weekly indemnity, accident and sickness	Quarterly
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AMERICAN BOARD EXAMINATIONS

Anesthesiology. Sec., Dr. Curtiss B. Hickcox, 80 Seymour St., Hartford 15, Conn. Oral, Coronado, Calif., Apr. 4-7, and Memphis, Oct. 14-17.

Dermatology and Syphilology. Sec., Dr. George M. Lewis, 66 East 66th St., New York 21. Written, various centers, Apr. 5. Oral, New York, June 8-10.

Internal Medicine. Sec., Dr. William A. Werrell, 1 West Main Street, Madison 3. Written, various centers, Oct. 15. Final date for filing applications is May 1. Oral on regional basis, Kansas physicians at St. Louis April 4-6 or 5-7.

Obstetrics and Gynecology. Sec., Dr. Paul Titus, 1015 Highland Bldg., Pittsburgh 6.

Ophthalmology. Sec., Dr. Edwin B. Dunphy, 56 Ivie Road, Cape Cottage, Maine. Oral, New York, May 31-June 5; Chicago, Oct. 8-13.

Otolaryngology. Sec., Dr. Dean M. Lierle, University Hospital, Iowa City. Oral, Richmond, Va., May 1-5.

Pathology. Sec., Dr. Robert A. Moore, 1402 S. Grand Blvd., St. Louis.

Pediatrics. Sec., Dr. John McK. Mitchell, 6 Cushman Road, Rosemont, Pa. Oral, Cincinnati, March 30-April 1; Atlantic City, May 5-7.

Physical Medicine and Rehabilitation. Sec., Dr. Robert L. Bennett, 30 N. Michigan Ave., Chicago. Parts I and II, Philadelphia, June 16-17.

Plastic Surgery. Sec., Dr. Bradford Cannon, 330 Dartmouth St., Boston.

Preventive Medicine and Public Health. Sec., Dr. Ernest L. Stebbins, 615 N. Wolfe St., Baltimore. Biloxi, Miss., April 24-25.

Proctology. Sec., Dr. Louis A. Buie, 102-110 Second Ave., S.W., Rochester, Minn. Kansas City, Minneapolis, Philadelphia and San Francisco, May 12.

Psychiatry and Neurology. Sec., Dr. Francis J. Braceland, 102-110 Second Ave., S.W., Rochester, Minn.

Radiology. Sec., Dr. B. R. Kirklin, 102-110 Second Ave., S.W., Rochester, Minn. Oral, Atlantic City, June 5-9.

Surgery. Sec., Dr. J. Stewart Rodman, 225 South 15th St., Philadelphia. Written, various centers, October, 1951. Final date for filing applications is July 1.

Thoracic Surgery. Sec., Dr. William M. Tuttle, 1151 Taylor Ave., Detroit. Oral, New York, April 19.

Urology. Sec., Dr. Harry Culver, 314 Corn Exchange Bldg., Minneapolis 15. Chicago, Feb. 9-13, 1952. Final date for filing applications is Sept. 1.

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THE KANSAS PRESS LOOKS AT MEDICINE

Who Opposes Socialized Medicine?

It is frequently argued that most of the opposition to either socialized medicine or government-controlled medicine originates within groups whose motives are entirely selfish, and who are completely without interest in the welfare of the masses of people.

That argument cannot withstand the light of fact. Current proposals for government medicine have met with the formal opposition of 15 state legislatures, and the amazing total of 10,000 national, state and local organizations. Among these are the General Federation of Women's clubs, the American Legion, the Veterans of Foreign Wars, the American Bar Association, and the American Farm Bureau Federation.

To say that organizations of this character are inspired by the selfish considerations of a few people is to say the ridiculous. They represent millions of individuals, of all political faiths, and of all the economic levels. They agree on the issue of government medicine for one reason—their belief that it would reduce the quality of medical care, that it would place a tremendous and unnecessary burden on the taxpayers, and that the growth of our established system of medical care, along with the many voluntary insurance plans, offers the best available solution to our health problems.

Those who would socialize, or otherwise regiment the practice of medicine, work on the theory that if a roof leaks, the solution is to burn down the house. That there are gaps in our medical care system goes without saying—and doctors are among the first to realize this, and to take measures to close them. In recent years, enormous progress has been made. The future will bring greater progress still—if the hand of the politician can be kept off the back of the doctor.—*Eureka Herald, October* 12, 1950.

* * *

Socialized Medicine

The New York Times said January 17: "One of the brightest spots in Federal activities during the past few years has been the high quality of medical service given veterans.

"...our veterans since 1945 have had medical care 'second to none.'"

The Federal Government, of course, owns veterans' hospitals and hires the doctors who treat the ex-servicemen.

That is genuine socialized medicine—and it has worked out all right in that case.

But the medical lobby, the American Medic Association (AMA), falsely labels national healt insurance as "socialized medicine," although privat hospitals would remain private under the nation health insurance program and private physician would keep their private practice.

Even so, in view of the fine record made by th real article—treatment for veterans—perhaps th AMA will quit trying to damn national health i surance as "socialism."

But we doubt it. "Socialism" is too good a cu word for the medical brass and its press agents t give up.—*Kansas City Labor Bulletin, February 1951.*

On an average weekday in February 1949 ther were about 4,569,000 persons from 14 to 64 years of age in the civilian population of the entire United States disabled by illness or some condition that prevented them from doing anything but occasionally part-time work. This does not include persons between these ages in resident institutions or in the armed services. A rough estimate for these two categories . . . brings the total up to 5,310,000, or 5.4 per cent of the population between these ages.—*Theodore D. Woolsey, Biostatistician, Public Health Reports, February 10, 1950.*

Accidental Death Rate High

Accidents killed 1,431 persons in Kansas during 1949, according to a report issued recently by the Kansas State Board of Health. That figure made accidents the fourth leading cause of death in this state for the year, with only heart disease, cancer and cerebral hemorrhage taking a higher toll. The Kansas rate was 20 per cent higher than that for the nation, and only six states had higher accidental death rates.

In the younger age group, from five years to 44, accidents are the leading cause of death. In the group between 15 and 24 years of age, accidents are responsible for one-half the total number of deaths.

In various forms—whether poliomyelitis, tuberculosis, cardiovascular disease, diabetes or any of the other abnormalities that are likely to alter a patient's social and economic activities — these chronic conditions account for three-fourths of all illness today. Progressive control of infectious diseases and the increasing numbers of elderly people in the population are bringing about a situation in which the chronic illnesses and preventive medicine may in the future demand the entire attention of physicians. — New England Journal of Medicine, August 10, 1950.

SMOOTHER CERVICAL CONIZATION

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Frequent necessity of cervical repair suggests the practicality of having a BLENDTOME ELECTROSURGICAL Unit in the office or clinic. With this instrument, the doctor is enabled to do a smoother cervical conization. The BLENDTOME cuts and coagulates simultaneously with a blended current. Scar and other tissue is cut through quickly and easily; blood and lymph vessels are almost instantly sealed. The cleaner field results in reduced trauma and operative shock, smoother convalescence and more rapid healing.

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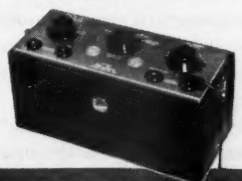

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ABSTRACTS FROM CURRENT LITERATURE

Neurologic Lesions in Infants

Neurologic Lesions in the Newly Born Infant. By Keith M. Haddow and Mildreda Norval, Ped., 6:2, Aug., 1950.

The authors' material covered the years 1944 through 1947. There were 4,411 deliveries on the obstetric service at St. Mary's Hospital, Rochester, Minnesota. Fifty-seven of these infants (1.3 per cent) had lesions, clinical or pathologic, which affected the nervous system primarily and these were evident during the first two weeks of life.

There were 12 infants who died during the first week which presented the following: anencephaly, exstrophy of the bladder, hydro cephalus and meningocele, anencephaly and prematurity, atelectasis, multible congenital anomalies, imperforate anus, polycystic kidney, prematurity, spina bifida, cerebral hemorrhage, asphyxia, external hydrocephalus, hypoplasia of the aqueduct of sylvius, cerebral anoxia, erythroblastosis fetalis.

Ten babies lived 18 days to eight months. Twenty-two infants survived for nine months or more, 13 had gross neurologic abnormalities, and two of the remaining nine were subjected to difficulty during birth. One had bilateral brachial palsy and one had two convulsive seizures. Both were normal at one year and 15 months of age. Six of the nine had muscle twitching and convulsions, but were well after intervals of seven months to four and one-half years. A remaining child had multiple congenital abnormalities and experienced convulsions.

A survey was made to determine the relationship of prolonged labor, asphyxia and delayed respiration to the occurrence of trauma and, thereby, neurologic lesions of the infant. As might be expected, prolonged labor appeared to increase the risk of intracranial hemorrhage and death of the infant. However, if the infants survived, there was little or no abnormality in subsequent development and no increase in neurologic disturbances, at least during the first few years of life. More children had convulsive disorders in the normal group than in those subject to prolonged delivery. The same held true among the infants who were asphyxiated; that is, there were more who had convulsive disorder in the control group than in those who were asphyxiated. There were more deaths and more abnormal infants among those who were asphyxiated, but these included a considerable number of premature infants, many not viable. If the infants survived the neonatal period, they developed normally, or at least no more abnormalities were noted among those who were asphyxiated than among the controls.

Anoxia caused by delay in respiration did not have a serious prognosis in those infants who survived the neonatal period. Even a delay of 11 to 15 minutes, while fatal in two instances, did not appear to cause any abnormality, at least during the first four years of life, in the infants who survive. —*D.R.D.*

* * *

Intravenous Quinidine in Ventricular Tachycardia

Intravenous Use of Quinidine in Ventricular Tachycardia. By A. Henry Clagett, Jr., Am. Jr. Med. Sc., 220:4, 281-287, Oct., 1950.

Doctor Clagett adds and compares his series of 13 patients to a list of 84 previously treated patients reported by 14 authors. He used quinidine lactate which is now commercially available, 0.65 grams in 10 cc. solution in ampoules.

The indications for intravenous quinidine were: 1. A critically ill patient, usually with ventricular tachycardia, in whom oral quinidine had been ineffective or whose condition was so serious that there was no time for trial of the drug by mouth. 2. Patients who were to have chest or heart surgery or procedures necessitating cardiac manipulation. In this group oral administration was given preoperatively and during surgery and afterwards the quinidine was added to infusion fluids.

When possible an oral test dose of 0.2 gm. was given first. After trying other procedures the one of choice was to dilute the 10 cc. solution containing 0.65 gm. quinidine lactate in 50 cc. of five per cent glucose in a 100 cc. flask and administering this at two cc. per minute with constant observation. If more drug was needed the procedure was repeated. The total dose needed varied from 0.4 to 3.25 grams.

The author emphasizes his belief that previous fatalities attributed to quinidine may have been the result of the disease, impurities, or too great concentration of the intravenous solution or its too rapid administration.

He concludes by saying he believes if quinidine lactate is given in the manner described it is relatively safe and may be lifesaving in patients developing ventricular tachycardia following myocardial infarction where oral quinidine could not be used or had failed, or where the patient's condition is so critical as to demand the greatest speed. Electrocardiograms and case summaries are included. The fact that several deaths occurred in this small series is evidence of the gravity of the cases selected. Autopsy evidence accounted for the deaths without putting blame on quinidine though quinidine deaths are discussed.—*P.W.M.*

In response to the Army's urgent call for 3,000 professional nurses, the American Nurses' Association recently established quotas for states to fill the requirements. Quotas were based on nurse population within each state. Kansas quotas were set at 25 for general duty, two for supervisory or head nurse duty, one nurse anesthetist, four operating room nurses, two psychiatric nurses—a total of 34.

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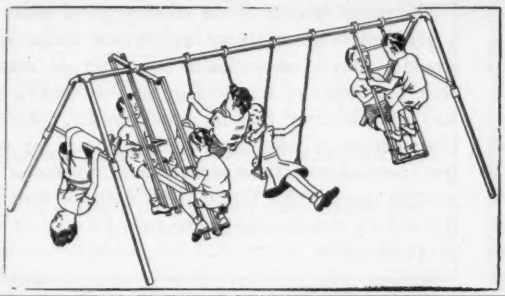

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A. B. BAKER, M.D., Professor of Neurology, University of Minnesota School of Medicine, Minneapolis.
RUSSELL N. DEJONG, M.D., Professor of Neurology, University of Michigan School of Medicine, Ann Arbor.
ANDREW B. JONES, M.D., Assistant Professor of Clinical Neurology, Washington University School of Medicine, St. Louis.
ROLAND P. MACKAY, M.D., Professor of Neurology, University of Illinois College of Medicine, Chicago.
JAMES B. CAMPBELL, M.D., Neurosurgeon, The Menninger Clinic, Topeka.
EDWARD C. WEIFORD, M.D., Neurosurgeon, Kansas City, Mo.

University of Kansas Faculty:

MARVIN L. BILLS, M.D., Assistant Professor of Psychiatry and Neurology.
DONALD F. COBURN, M.D., Associate in Surgery (Neurosurgery).
E. GREY DIMOND, M.D., Assistant Professor of Medicine.
B. LANDIS ELLIOTT, M.D., Assistant Professor of Psychiatry and Neurology.
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DONALD L. ROSE, M.D., Associate Professor of Physical Medicine.
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ELLSWORTH H. TROWBRIDGE, Jr., M.D., Associate in Psychiatry and Neurology.
HENRY C. TRACY, Ph.D., Professor Emeritus of Anatomy.
HERBERT A. WENNER, M.D., Assistant Professor of Pediatrics and Bacteriology.

SUBJECTS TO BE DISCUSSED

THE SCOPE OF NEUROLOGY AS APPLIED TO MEDICINE.
A REVIEW OF NEUROANATOMY.
THE EXAMINATION OF THE CEREBROSPINAL FLUID.
DIAGNOSIS OF ENCEPHALITIS AND POLIOMYELITIS.
NEUROLOGIC HISTORY AND EXAMINATION.
THE BLOOD SUPPLY OF THE BRAIN.
THE BLOOD SUPPLY OF THE BRAIN AND SPINAL CORD—Clinical Application.
DIAGNOSIS AND TREATMENT OF TIC DOULOUREUX.
HEADACHE AND MIGRAINE.
DIAGNOSIS AND TREATMENT OF MENINGITIS.
PRINCIPLES OF CEREBRAL LOCALIZATION.
SYRINGOMYELIA.
MONONEURITIS AND POLYNEURITIS.
INTRACRANIAL TUMORS.
PARKINSON'S DISEASE AND PARKINSONISM — Diagnosis and Treatment.
MULTIPLE SCLEROSIS.
FUNDUS CHANGES IN NEUROLOGIC DISEASES.
THE MENTAL DEFICIENCY PROBLEM (Neurological).
THE MANAGEMENT OF HEAD INJURIES.
DIAGNOSIS AND TREATMENT OF NEUROSYPHILIS.
THE DIFFERENTIAL DIAGNOSIS AND TREATMENT OF CORD TUMORS AND INTERVERTEBRAL DISC.
SPINAL CORD LOCALIZATION AND DIAGNOSIS.
DIAGNOSIS AND TREATMENT OF CONVULSIVE DISORDERS.
VASCULAR DISEASE OF THE BRAIN AND SPINAL CORD

Panel Discussion:

(a) Medical Aspects
(b) Neurologic Diagnosis
(c) Medical Treatment
(d) Rehabilitation

Program will also include two Neurological Clinics, both of two hours' duration and both including case presentation.

Tracheobronchial Aspiration

Tracheobronchial Aspiration with a Urethral Catheter. By Leonard Cardon, J.A.M.A., 142:4, 1039-1044, Apr. 8, 1950.

The author points out that a frequent though often unrecognized complication of many diseases seen in daily practice is obstruction of the airway by accumulating tracheobronchial secretion with resultant asphyxia. This is not alone an anesthetic, postoperative, post-traumatic or neonatal problem but one encountered often in medical disease. He mentions bulbar polio, tetanus, whooping cough, pneumonia, acute cerebral damage, chest injury, acute pulmonary edema, morphine depression, barbiturate poisoning, acute alcoholism, diabetic or uremic coma, etc.

He recognizes that in certain cases bronchoscopy by a trained bronchoscopist is necessary, but points out that the greater number can be well cared for by repeated aspiration by means of urethral catheter introduced through the nostril and into the trachea and upper bronchial tree. The technic described is simple and one that any doctor can master with a little practice and patience. The value of repeated aspirations, attempting to keep the tracheobronchial tree free of mucus, is stressed. Using the catheter technic repeated aspirations at frequent intervals can be done without fear of trauma.

The following contraindications are listed: 1. Pronounced local disease of larynx; 2. Marked glottic spasm which calls for bronchoscopy or insertion of a Magill tube; 3. Obstruction or gross purulent infection of nasal passage—here the catheter may be inserted through the mouth. 4. Severe coughing, straining or struggling provoked by attempted insertion of catheter in a patient in whom increased intracranial pressure may be dangerous, i.e. cerebral hemorrhage, brain tumor. 5. If catheter aspiration proves ineffectual in removing obstruction, bronchoscopy and aspiration under direct vision is indicated.

Asphyxia by accumulating tracheobronchial secretion is a frequent complication and may cause symptoms and death in many common medical diseases and emergencies. Early recognition and frequent, adequate tracheobronchial aspirations will save many lives.

Ten cases illustrating the immediate life-saving effect of this procedure are reported.—*H.F.S.*

* * *

Salivary Gland Tumors

Salivary Gland Tumors of the Submaxillary Gland Associated with Calculi. By Ephraim L. Manning and Maurice A. Michael, Ann. O., R., L., 59, 141-145, Mar., 1950.

On diagnosing a neck swelling as an affection of the submaxillary gland, we first think of an obstructing calculus or an infection, or both, and usually dismiss the diagnosis of tumor quickly, especially if a calculus can be demonstrated by palpation and x-ray examination. However one must consider salivary gland tumor (so-called mixed tumor) of the submaxillary gland even if calculi can be demonstrated.

The term "mixed tumor" applied to these neoplasms was originally proposed by Minssen in 187[illegible], but they are not teratomas since teratomas possess definite tissues forming atypical structures and rudimentary organs. These neoplasms are generally believed to be derived from epithelium and thus are not "mixed tumors."

The main element of the salivary gland tumor is a polymorphous epithelial cell which forms into strands, alveoli and tubular structures or masses closely arranged or scattered in the surrounding tissue. The cell itself may be round, stellate, cuboidal, columnar or spindle shaped. Other than the cell, the most characteristic structure is an excess of mucoid tissue which intermingles with the supporting stroma and is believed to be a product of the neoplastic cells.

Salivary gland tumors occur at all ages, with an average age of onset of apparently 35 to 40 years, and last on the average about six to nine years. They occur about equally in males and females, and in the salivary glands occur chiefly in the parotid gland (90.6 per cent with, in order of frequency, next the submaxillary (8.8 per cent), and then sublingual gland (0.6 per cent). However, they also appear in the palate, cheek, lips, tongue, pharynx, and nares.

The usual history is of a previously quiescent nodule in or over the periphery of one of the larger salivary glands, growing slowly, taking about five years to reach the size of a walnut and about 10 years to reach the size of a lemon. These tumors have a marked tendency to recur after operation because of the difficulty of removing all portions of the tumor, especially in the parotid gland. Malignant types of these tumors do occur and invade adjacent structures, and there have been many cases of metastasizing mixed tumors, although this occurs infrequently.

Salivary gland tumors are resistant to irradiation and surgical excision is the accepted treatment.

Calculi are frequently found in glandular tumors due to stasis of secretions and it might be expected that if a salivary gland tumor should be found with calculi it would be due to this cause.—*E.L.G.*

Annual meeting, Kansas Medical Society, May 14-17, 1951, Topeka.

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Basic Principles in General Surgery, Two Weeks, starting April 2.
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Cerebral Palsy, Two Weeks, starting July 9.
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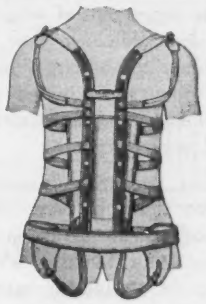

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BOOK REVIEWS

The Auditory Clinic. By Moe Bergman. Published by Audiology Foundation, Chicago. 107 pages, 12 illustrations. Price $1.00.

This monograph is presented as a guide and contains suggestions of ways and means for organizing an ideal audiology clinic, in which the program of auditory rehabilitation includes the cooperation of the otologist, psychiatrist, psychologist, social worker, audiologist and instructors of speech, speech reading and auditory training.

The Veterans Administration Auditory Rehabilitation Clinic in New York has been used as the "pilot" unit for the book. Details of construction are offered for building sound proof testing rooms. Lists of equipment essential for testing hearing through pure tone, speech, masking and galvanic skin response are given, with suggestions for assembly.

Brief discussions were presented on the personnel of the audiology clinic, duties of each person, the continual need for complete cooperation of the staff, and re-evaluation of the daily programs. True interest in the individual was stressed, and repeated emphasis was made on the need for detailed planning and study of the instructional program, of the area to be serviced, and of the types of programs to be presented, as well as of plans for architectural acoustics.

The author did not attempt to present specific communicative skills, techniques for testing hearing, or selection of hearing aids. He has presented organized information for planning a clinic for the rehabilitation of the acoustically handicapped.—*B.J.M.*

The Antihistamines—Their Clinical Application. By Samuel M. Feinberg, M.D., Saul Malkiel, M.D., and Alan R. Feinberg, M.D. Published by Year Book Publishers, Inc., Chicago. 291 pages, 27 illustrations. Price $4.00.

This timely volume is divided into two sections, the first embracing experimental studies and the second clinical observations. The authors have further divided the book into 10 parts, the first five covering the experimental data on histamine, the chemistry, pharmacology, and bioassay in man of the antihistamines, and anaphylaxis. The next five parts relate to clinical studies of respiratory allergy, dermatoses, miscellaneous manifestations, including collagen diseases, tuberculosis, motion sickness, the common cold, and radiation sickness, and finally the administration, dosage and toxicology of the antihistaminics. An appendix lists the proprietary antihistaminic preparations.

The first section reflects adequately the background for experimental work in the field, but is more an introduction than a reference for research. The second section is marked by conservatism in the reports of the authors. They conclude that the therapeutic usefulness of the antihistamines is predominantly in the control of sneezing and the rhinorrhea of hay fever. They stress the limitations of the antihistamines and discourage their use in the treatment of asthma, and as the sole agents in the control of the allergic syndrome. The over-the-counter sale of the less potent drugs is condemned.

This volume is well organized and presented, and includes an imposing bibliography of 586 items. The opinions stated are confirmed and well documented. The application of the material included in this book should be invaluable to the clinician and should perhaps curtail the indiscriminate use and abuse of the antihistamines in the hospital and in general practice.—*D.H.D.*

Mental Health Grants to Kansas

Three Kansas institutions were awarded grants which total $78,175 for mental health training for psychiatrists, psychologists, nurses, social workers and medical students, according to a recent announcement from the Federal Security Agency. The funds are part of a total of $1,915,708 allocated for the purpose by the Public Health Service, National Institute of Mental Health.

The University of Kansas will receive $31,592 for clinical psychology, psychiatric social work and undergraduate psychiatry, the Menninger Foundation, Topeka, was awarded $36,927 for graduate psychiatry and clinical psychology, and the Wichita Guidance Center was granted $9,656 for clinical psychology.

The mental health training program was initiated in 1947 and was expanded in 1949. Training grants have been given to institutions in 32 states.

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Relationship of Stress to Autonomic Lability

Studies in psychosomatics have shown that functional disorders often are a result of the patient's inability to adjust to emotionally stressful situations (stressor factors).

Nervous tension and chronic anxiety, discharged through a labile Autonomic Nervous System, can cause somatic disturbance. [1,2] Such states may involve any one of the organ systems or several at one time. [1,3] The outline below is designed to relate gastrointestinal and cardiovascular symptomatology to the exaggerated response of the autonomic nervous system.

	Physiologic Effects of Autonomic Discharge	
	Sympathetic	Parasympathetic
Gastro-intestinal System	Hypomotility Intestinal Atony Hyposecretion Reduced salivation	Hypermotility Gastrointestinal spasm Hypersecretion
Cardio-vascular System	Rapid heart rate Peripheral vaso-constriction	Slow heart rate Vasodilatation
Functional Manifesta-tions	Palpitation Tachycardia Elevated blood pressure Dry mouth and throat	Heartburn Nausea-vomiting Low blood pressure Colonic spasm

The data here tabulated is from references 3,4,5,6,7, given below.

When the clinical picture is suggestive of functional disorder, the diagnosis is supported by the presence of the following indications of autonomic lability:

Variable Blood Pressure
Body Temperature Variations
Changing pulse rate
Deviations in B. M. R.
Exaggerated Cold Pressure Reflex
Oculo-Cardiac Reflex Abnormalities
Glucose Tolerance Alterations

Therapy in these cases is directed toward: 1) relieving the somatic disturbance to prepare the patient for psychotherapy*; 2) guidance in making adjustment to stressful situations and correction of unhealthy attitudes.

*Drug treatment using adrenergic and cholinergic blocking agents in conjunction with sedatives. 8,9,10.

1. Ebaugh, F.: Postgrad. Med. *4:* 208, 1948. 2. Wilbur, D.: J.A.M.A. *141:* 1199, 1949. 3. Williams, E. and Carmichael, C.: J. Nat'l. Med. Assoc. *42:* 32, 1950. 4. Goodman, L. and Gilman, A.: The Pharmacological Basis of Therapeutics, The Macmillan Co., 1941. 5. Katz, L. et al: Ann. Int. Med. *27:* 261, 1947. 6. Weiss, E. et al: Am. J. Psychiat. *107:* 264, 1950. 7. Alvarez, W.: Chicago Med. Soc. Bulletin, 581, 1950. 8. Rakoff, A.: A Course in Practical Therapeutics, Williams and Wilkins, 1948. 9. Karnosh, L. and Zucker, E.: A Handbook of Psychiatry. C. V. Mosby Co., 1945. 10. Harris, L.: Canad. M.A.J. *58:* 251, 1948.

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State House,
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Rickets cannot enter . . . .

. . . where the SUN never sets

The infant or child who receives Mead's Oleum Percomorphum daily is assured of his supply of vitamin D.

No matter what the clime or season, he lives "in a land where the sun never sets," protected from the shadow of rickets.

Supplying 60,000 vitamin A units and 8,500 vitamin D units per gram, Mead's Oleum Percomorphum provides this dependable prophylaxis in small doses at low cost.

Mead's Oleum Percomorphum comes in bottles of 10 and 50 cc., and of 50 and 250 capsules. Patients appreciate the economy of the large sizes.

Mead's Oleum Percomorphum is the *original* Oleum Percomorphum product—with a background of 17 years of quality and clinical acceptance. Available in all drugstores.

When you prescribe Oleum Percomorphum
Specify

MEAD'S

Oleum Percomorphum

MEAD JOHNSON & CO.
EVANSVILLE 21, IND., U.S.A.